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SURGICAL TREATMENT OF CHRONIC CONSTRICTIVE PERICARDITIS

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Chronic constrictive pericarditis long has been recognized as a clinical entity. Galen knew that pericardial effusions and scirrhous thickening of the pericardium occurred in certain animals and supposed that the same conditions might affect man and interfere with proper cardiac function.* Lower is credited with the first satisfactory description of human chronic constrictive pericarditis in 1669. The syndrome became better understood as the result of the observations of such authors as Pick, Griesinger, Kussmaul, Chevers and Wilks. It was first recognized as a surgical problem by Weill in 1896 and Delorme repeatedly pointed out from 1895 to 1898 that relief of symptoms might be expected from pericardial resection. This procedure finally was carried out in 1913 by Sauerbruch and by Rehn. It first was performed in this country in 1928 by Churchill.

With the application of surgical methods for the cure of this condition there arose an interest in better understanding the precise underlying physiopathology. Much valuable information has been obtained by carefully comparing the result of treatment with the anatomic situation encountered at operation and the procedure executed, by cardiac catheterizations performed before and after operation in successfully and unsuccessfully treated patients, and by experimental studies in animals. In spite of the fact that broad general agreement has been reached, a number of problems have remained debatable. Among the questions concerning which disagreement still exists are the following: When should operative treatment be carried out—as soon as the diagnosis is established or only after incapacitating symptoms are present? Can operation be performed with

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* An excellent historical review is found in P. D. White's St. Cyres Lecture.[15]

reasonable safety in cases of active tuberculous constrictive pericarditis or should it be deferred until signs of activity are no longer present? Over what chambers of the heart is compression most important in bringing about the signs and symptoms of this disorder? Decompression of what portions of the heart is most essential in obtaining a satisfactory postoperative result? How extensive a resection should be attempted? What operative approach provides the best and safest exposure? Are all imperfect results due to inadequate decortication? It is the purpose of this communication to attempt to answer such questions from personal experiences and from a survey of the clinical and experimental literature.

CASE REPORTS

All patients were studied carefully for evidence of tuberculosis, rheumatic fever and valvular heart disease. Liver function tests and electrolyte studies were made as a routine. All patients were prepared for operation as well as could be by salt restriction and diuretics, in some by digitalization, and in one instance by use of carboresins. In the first patient operative exposure was obtained by transecting the sternum at the level of the third interspace and splitting it below down through the xiphoid. In the fourth patient a third right anterior intercostal incision was used. In the remainder the entire sternum was split longitudinally.

Case 1. The patient was a 56 year old white man who had been treated erroneously for several years for cirrhosis of the liver. He had had a persistently enlarged liver, ascites, and edema of the lower extremities. More recently he had suffered from dyspnea. When admitted to the hospital in April 1948 his heart was moderately enlarged, the heart sounds were faint, the liver edge was palpable about 20 centimeters below the costal margin; the abdomen was distended with ascitic fluid and the lower extremities were edematous. Repeated venous pressure determinations revealed values of 24 centimeters or greater. Total serum proteins were 4.35 grams per cent, the albumin fraction being 1.41, the globulin 2.94.

Operation was performed on April 29. The pericardium was very thick and adherent over the entire surface of the heart. The pericardium over the ventricles was removed anteriorly and back beyond the mid-portion on both sides. Most of it was excised from over the right atrium and a portion from over the left atrium. The excised tissue showed extensive nonspecific fibrosis.

During the six years since treatment the patient has led an active life and has remained entirely well.

Comment. This case illustrates a common error in diagnosis; the futility of treating patients with chronic constrictive pericarditis for cirrhosis of the liver; and the gratifying relief of symptoms which usually follows proper operative treatment.

Case 2. The patient was a white man aged 49 who was admitted to the hospital on Oct. 20, 1948, with the history of severe dyspnea, orthopnea, ascites, and edema of the lower extremities of one year's duration. He became much worse three months before admission. Considerable, but only transient, relief followed a pericardial tap. He had been treated over a long period of time with streptomycin.

He was emaciated, mildly orthopneic and slightly cyanotic. He had poor heart sounds; a venous pressure of 27 centimeters; a pulsus paradoxus; shifting dullness in the abdomen

without massive ascites; edema, and a liver which extended 10 centimeters below the right costal margin. Serum proteins were 7.20 grams per cent, the albumen fraction being 3.79, globulin 3.41. Cephalin flocculation was 3 plus. Electrocardiograms revealed flat T waves and very low voltage. Pericardial paracentesis was done on several occasions. Radiologic studies after aspiration of pericardial fluid and injection of air showed marked thickening of the pericardium. Following the first pericardial tap there occurred a bilateral pleural effusion. The heart itself was not enlarged and it exhibited poor pulsation. The aspirated fluid contained acid fast bacilli. Streptomycin therapy was continued. During the first few days there was a low grade fever and leukocytosis. Thereafter the temperature and leukocyte count were normal. The blood pressure ranged from 90/75 to 105/90. Operation was deferred in the hope that his active tuberculous process could be brought under control. The last pericardial tap on November 12 yielded fluid still positive for acid fast organisms. He became progressively more cyanotic and orthopneic. By the time he was finally operated upon on November 30 he was in a desperate condition, breathing with great difficulty and markedly cyanotic while sitting straight up in an oxygen tent.

An extensive pericardectomy was done. The pericardium was markedly thickened over the entire heart. There was no calcification. He remained deeply cyanotic throughout the procedure in spite of vigorous assisting of respiration with high oxygen content mixture. During the closure of the sternal wound generalized convulsions took place, followed rapidly by cardiac stand still. Resuscitative efforts were fruitless.

The pericardium showed evidence of tuberculous inflammation with extensive fibrosis. There was tuberculosis of the lungs, liver and spleen.

Comment. This case illustrates the difficulties which may follow a policy of trying to defer operative treatment until an active tuberculous process becomes quiescent. During the period of observation the patient's condition became progressively worse and operation had to be done eventually as a desperate last resort. One cannot help believing that earlier in the course of his illness the patient might have tolerated pericardiectomy and have been relieved of his cardiac symptoms. Whether or not he would have then conquered his tuberculous infection is problematic.

Case 3. The patient was a 53 year old white woman who was admitted to the hospital May 4, 1949, because of dyspnea, orthopnea, abdominal swelling, and peripheral edema for two weeks, and a varicose ulcer of the right leg for the past 15 years.

She had a quiet heart, auricular fibrillation, paradoxical pulse, dullness at both bases, ascites, marked peripheral edema and an enlarged liver which extended about 7 centimeters below the right costal margin. The blood pressure was 120/80—venous pressure was 31 centimeters. Fluoroscopic and roentgenographic studies showed poor cardiac pulsations, calcification of the pericardium, and pleural effusion on the right side. Electrocardiograms showed auricular fibrillation, flat T waves, and low voltage. The patient's condition improved on sodium restriction, mercurial diuretics, and digitalis therapy, and she was discharged on May 29.

She was readmitted July 12 with recurrence of the same complaints. On the same medical regimen she improved and lost 43 pounds. She was operated upon on August 30. The pericardium was thickened and adherent everywhere but was least involved over the anterior aspect of the right ventricle. The calcification was greatest in the posterior half of the pericardium. A large calcific plaque was adherent to the right atrium and a broad constricting band of calcified tissue extended posteriorly towards the ostia of the vena cavae. It finally was freed and divided with release of obvious constriction. The anterior ½ to ⅔ of the pericardium was excised. The tissue removed showed nonspecific pericarditis with calcification.

Convalescence was uneventful. At the time of discharge the venous pressure was 16 to

18 centimeters. She was advised to take digitalis and to remain on a low salt diet. It soon became apparent, however, that she followed instructions poorly. She obviously was mentally retarded. During the four and three-quarter years since operation she has been relatively well but has not had normal health. She has been seen at frequent intervals. She was free of complaints until June 1950 when she developed mild exertional dyspnea. In February 1952 she was in the hospital for a week in cardiac failure. It was obvious she had not adhered to her diet and with salt restriction and diuretics she rapidly lost her symptoms and 32 pounds of weight. She was readmitted in November 1952 and again in March 1953 for treatment of varicose veins and the ulcer of the leg. She seemed free of cardiac signs and symptoms.

Although she had no complaints when seen on Jan. 15, 1954, her venous pressure was 26 centimeters, whereas most venous pressure determinations during the preceding years had been in the neighborhood of 17 centimeters. She was admitted to the hospital on January 21 for consideration of reoperation. Again she lost weight—13 pounds—with nothing more than salt restriction. She had auricular fibrillation, as she had had throughout the period of observation at this institution. Fluoroscopic study revealed the cardiac contractions to be active. The heart was enlarged generally and the left atrium was enlarged in particular. Venous pressure was 15 centimeters. Since she had remained relatively well over a period of years in spite of failure to adhere to a proper dietary regimen, and her rare bouts of difficulty were quickly resolved by sodium restriction, it was thought that operation was not indicated and that further efforts should be made to have her follow better the medical advice offered.

Comment. This patient has done reasonably well over a period of years although on occasions she has developed difficulties which were quickly resolved by sodium restriction. She has continued to have auricular fibrillation. She has cardiac enlargement. It is not known whether her residual difficulties are due to some persistent pericardial restriction or to heart disease. Fluoroscopically cardiac contractions are vigorous. Since she can be kept in good health by sodium restriction reoperation has not been advised.

Case 4. The patient was a 54 year old man who was first admitted to the hospital on May 11, 1950, with a history of dyspnea, orthopnea and engorgement of the neck of five or six week's duration. He had obvious evidence of superior vena caval obstruction and of hypertensive cardiovascular disease with congestive failure. The blood pressure was 210/120. Venous pressure was 58 centimeters in one arm, and 25 centimeters in one ankle. Study of a surgically removed supraclavicular node showed lymphoid hyperplasia. Roentgenologic studies revealed marked cardiac enlargement, an elongated and tortuous thoracic aorta, and pleural fluid. Gastrointestinal barium studies and intravenous and retrograde pyelograms were not remarkable. Angiocardiograms showed reflux of the opaque medium into the azygous vein and into several tributaries of the subclavian and innominate veins. Electrocardiograms demonstrated left ventricular hypertrophy and, on one occasion, auricular fibrillation. He was discharged on June 12 and readmitted on November 14.

The blood pressure was 230/145. The venous pressures in the arms were 27 and 25.5 centimeters; in the ankles 14 and 16.5 centimeters. Cardiac catheterization was done on December 1. The catheter met a definite obstruction just at the point of entry of the superior vena cava into the atrium. Within the stenotic area the pressure was 15.8 to 12.4 mm. mercury, and just beyond in the right atrium 9.9 to 5 mm. The patient was discharged and readmitted on December 24. The cause of the vena caval obstruction was not known.

On January 10 exploration was done through an anterior right intercostal incision. A firm mass could be felt posterolateral to a dilated aorta in the region of the superior vena cava. The pericardium in general was not particularly thickened and ventricular pulsations were seen and felt to be vigorous. Near the base of the heart the pericardium seemed thicker

and adherent. A large portion of the pericardium was excised. It was everywhere adherent to the heart and great vessels. It was grossly thickened only near the origin of the aorta and superior vena cava. The aorta was dilated to a diameter of 7 centimeters. After the pericardium was removed one could feel a strong hard mass of calcium which appeared to form a ring about, and to be incorporated in, the superior vena caval ostium. No other calcium could be demonstrated in the atrial wall and there seemed to be no obstruction of pulmonary venous return. Examination of the excised pericardium showed nonspecific pericarditis.

The patient was not improved. He died on July 21, 1951. Nothing is known of the final period of his illness.

Comment. Although this patient had generalized adherent pericarditis it appeared to cause no general constriction of the heart, but was associated with a calcific stenosis of the superior vena caval ostium. Operative pericardiectomy consequently was ineffectual in relieving the superior vena caval obstruction. Had the patient not been in dire straits from hypertensive cardiovascular disease it might have been advisable to attempt to bypass the obstruction. It seems clear that he died not of superior vena caval obstruction, but of cardiac failure.

Case 5. This patient was first admitted to the hospital July 25, 1951, at the age of 15. A diagnosis of pulmonary tuberculosis had been made in January 1950 and he had been treated in a sanitarium for 17 months. At the time of admission he apparently had hepatomegaly, ascites, dyspnea, and mild orthopnea. During the last 10 months of his stay in the sanitarium roentgenograms had revealed no evidence of active pulmonary tuberculosis and sputum cultures and smears had been negative. He still could not lie flat but required two pillows, had some peripheral edema, and even at bedrest the slightest activity caused dyspnea. Digitalis therapy did not have any appreciable effect. A diagnosis of chronic constrictive pericarditis was made.

When admitted to the hospital he had edema of the lower extremities but only suggestive evidence of ascites. The liver edge was 8 centimeters below the right costal margin. The heart sounds were quiet, the blood pressure 95/50, and the venous pressure 35 centimeters. Fluoroscopic and roentgenographic studies showed poor cardiac pulsations, extensive pericardial calcification, and mild bilateral pleural effusion. Cardiac catheterization revealed high pressures in the venae cavae, right ventricular hypertension and an elevated right ventricular diastolic pressure with a mid-diastolic plateau. With diuretics the patient's weight dropped from 98 to 90 pounds.

Pericardiectomy was done on July 28. The pericardium was greatly thickened everywhere and was calcified. There were plaques of calcium in the heart itself near the apex of the ventricles anteriorly and at the bases to the right of the point of origin of the aorta. The pericardium was removed anteriorly from the entire heart and posteriorly back beyond the mid-portion of the heart on both sides. The venous pressure, which was 34 centimeters at the beginning, was 26.5 centimeters at the completion of the procedure. The weight at the time of discharge was 81 pounds.

When seen on October 22 he seemed much improved but was not entirely well. He could lie flat with comfort and was attending school a few hours a day. His liver was a little smaller. However, he still had definite through minimal ankle edema and a venous pressure of 24.5 centimeters.

He was readmitted on March 17, 1952. Although he said he felt much better, he had occasionally required mercurial diuretics for mild peripheral edema and at times had noted dyspnea with mild exertion. Cardiac catheterization was repeated. On the first occasion before operation right atrial pressure was 30/21. It was now 18/11. Ventricular pressure had been 57 to 53/20 to 14. It was now 75 to 45/19 to 6. The pressure tracing showed a mid-diastolic plateau. The left femoral artery pressures were 117 to 106/71 to 62.

He was operated upon on March 22 through a left anterolateral thoracotomy. The orig-

inal resection of pericardium on the left proved to have been so extensive it was feared additional resection might contribute little towards improving the cardiac function. However, the remaining pericardium was excised back to the posterolateral border to the heart. The myocardium seemed thin and flabby. At the completion of the procedure, the venous pressure had risen to 60 centimeters and his condition seemed precarious. Rales were audible throughout both lungs. Marked improvement followed phlebotomy—740 cc. of blood being withdrawn. His convalescence was then rapid and he was discharged on April 6.

Examination of the excised pericardium showed only marked nonspecific fibrosis with calcification, as had been true of that removed at the first operation. Neither specimen revealed evidence of tuberculosis.

During the two years which have elapsed he has remained entirely well and has led an active life. In January 1954 he weighed 122 pounds and had no edema. His liver was not enlarged, heart sounds were good, the blood pressure 130/70 and venous pressure was 10.5 centimeters.

Comment. This case has several points of interest. Although the first extensive pericardial resection was followed by obvious improvement, the patient was not well. Repeat cardiac catheterization revealed the vena caval and right atrial pressures to be significantly lower than before surgery, although still elevated, and the right ventricular hypertension still to be present. Surgical removal of the posterolateral remnant of pericardium—although it seemed at operation to be relatively inconsequential—brought about restoration to health. The exposed ventricular myocardium seemed thin without good tone and the severe transient cardiac failure which immediately followed operation logically may have been related to impaired myocardial function. The ultimate complete recovery illustrates the tremendous recuperative power of such apparently weakened heart muscle. The failure to demonstrate any histologic stigma of tuberculosis in the excised pericardium in a patient so apparently originally of tuberculous origin is noteworthy.

Case 6. The patient was a 62 year old white man who first was admitted to the hospital on July 29, 1951. Thirty-eight years before he had noted transient ankle edema following an accident in a coal mine and had been told he had heart disease and a poor prognosis. He recovered quickly and had no cardiac symptoms until 10 months before admission when he began to have dyspnea and ankle edema. He was placed on a low salt diet and digitalis. The following month he was admitted to another university hospital where, after careful study, the conclusion was reached that his difficulty was due to mitral stenosis and insufficiency and not to constrictive pericarditis. Calcification of the pericardium was evident on roentgenographic examination. Several months later he was admitted to another hospital in frank heart failure with massive ascites, peripheral edema, dyspnea, and orthopnea. He was treated by paracenteses, sodium restriction, and carboresins with some slight improvement. At that time the venous pressure was 22 centimeters. He had auricular fibrillation. Total proteins ranged from 5.6 to 6.15 grams per cent, the albumen fraction from 3.26 to 3.58.

At the time of his admission the findings were much the same. There were systolic and diastolic apical murmurs. The venous pressure was 19 centimeters. By fluoroscopic and roentgenographic study both ventricles and the left atrium were found to be enlarged. Calcification was noted in the region of the mitral valve. There was poor cardiac pulsations and the pericardium was calcified. Cardiac catheterization revealed pressures in superior and inferior venae cavae and right atrium all to be about 20/15 mm. of mercury. The catheter was not passed into the right ventricle. It was the impression of the medical service that his difficulties were the result of his mitral valvular disease.

He was readmitted on May 15, 1952. He had been somewhat better on his low sodium diet and carboresin therapy. Formerly he had required paracenteses every three weeks, but for the past 11 months only once every three or four months. The blood pressure was 150/82, and the venous pressure was 28 centimeters. The murmurs were unchanged. Electrocardiograms revealed auricular fibrillation, premature ventricular contractions from different foci and evidence of right ventricular hypertrophy. The decision was made to do a pericardiectomy and he was readmitted on May 21 for this purpose.

Operation was performed on May 22. The pericardium was thick and adherent everywhere and in large part calcified. The anterior ⅔ of it were removed from over the entire heart. There was a remarkably tight calcific band beginning in the right atrioventricular groove and extending across the left atrioventricular groove. Its mid-portion could be divided and excised with rongeurs, with obvious relief of considerable constriction. The same calcific plaque extended back to the region of the vena caval ostium but it seemed here to involve the atrial wall and could not be removed. Examination of the excised tissue revealed evidence of healed nonspecific pericarditis with calcification. Convalescence was uneventful.

The patient has done very well altogether during the two years since operation. He has led a fairly active life. He has required no paracentesis. Venous pressures have been relatively normal, ranging from 7.3 to 16.5 centimeters. He has continued on a low sodium diet and carboresins. Auricular fibrillation has persisted. Until October 1953 he received digitalis and up until that time occasional intramuscularly administered mercurial diuretics. Since then he has taken neohydrin about once weekly. His liver is barely palpable. Although he is not entirely well he is much pleased with his state of health.

Comment. This patient has classic auscultatory and roentgenographic signs of mitral heart disease. Operative treatment was deferred for a long time in the belief that the pericardial calcification was of relatively little importance. Pericardiectomy has brought about remarkable improvement. It is believed that the residual symptoms are attributable to his known mitral valvular disease. The patient has not been advised to have further surgery either to relieve his mitral stenosis or to carry out more extensive pericardial resection. He is well pleased with the result of his treatment.

Case 7. This patient was a 19 year old white man who was admitted to the hospital on May 31, 1952, with a history of dyspnea and edema of the lower extremities of five years' duration. He had long been thought to have cirrhosis of the liver, and a punch biopsy of the liver obtained in 1949 was said to have confirmed this impression. His symptoms had grown progressively worse and ascites had developed.

He had ascites, massive edema of the legs, a quiet heart, pulsus paradoxus, and a liver which extended about 7 or 8 centimeters below the right costal margin. The blood pressure was 90/70 and the venous pressure was 21 centimeters. There was a faint calcification of the pericardium on roentgenographic examination. Cardiac catheterization revealed elevated atrial pressure (16 to 12/10 to 8) and normal right ventricular systolic pressure (22/5) with a mid-diastolic plateau. Pulmonary artery pressure was 28/1. Following a transient bout of auricular fibrillation which disappeared with digitalization he was operated upon on June 9.

The pericardium was everywhere very thick and adherent. There were scattered flecks of calcium. Resection was complete over the ventricles and atria anteriorly, back along the vena cavae on the right and beyond the level of the phrenic on the left. The excised tissue showed evidence of nonspecific pericarditis.

Convalescence was uneventful. By the time of discharge 12 days after operation he had lost 21 pounds. His venous pressure was 13 centimeters. When seen in April 1953 he was entirely well. He had gained 30 pounds and had no edema. He was leading a normal and active life. He has remained entirely well. In March 1954 his blood pressure was 130/80 and the venous pressure was 16 centimeters. The liver edge was not palpable.

Comment. This is an excellent result in a young man with symptoms of five years' duration, long treated erroneously as a case of cirrhosis of the liver.

Case 8. The patient was a 53 year old white man who was admitted to the hospital on Jan. 27, 1953. Exertional dyspnea had begun 13 months before admission and he had been forced to quit work five months later. By June 1953 he had a large left pleural effusion and by October massive swelling of abdomen, arms, and legs. His venous pressure is reported to have been 30.5 centimeters at that time. He had been treated with no success by sodium restriction, diuretics, crystodigin and restricted water intake.

On admission he had massive edema of the entire body, ascites, large bilateral pleural effusions and a quiet heart. Blood pressure was 100/80, and the venous pressure was 26 centimeters. Total proteins were 4.5 grams per cent; albumin 3.26 grams. He had characteristic fluoroscopic and roentgenographic findings and abnormal ST-T and QRS complexes with extremely low voltage. He was continued on a low salt diet. No diuresis followed use of mercurial diuretics.

Operation was performed on February 2. Beforehand 1800 cc. of fluid was removed from the right pleural cavity and the same quantity from the left. The pericardium was everywhere thickened and adherent. There were small flecks of calcium. The pericardium was removed from the anterior surface of the heart and back at least to the midportion laterally on each side. The excised specimen showed nonspecific chronic pericarditis.

He lost weight rapidly and, when diuretics were given, excellent diuresis occurred. By the time he left the hospital three weeks after operation he had lost 63 pounds of water.

He has remained entirely well; is working; taking a normal diet and no medication.

Comment. This is an excellent result in a severely incapacitated individual. The failure to control edema with strict medical regimen and the rapid loss of fluid after operation are noteworthy.

Case 9. The patient was a 20 year old white man who was admitted to the hospital on May 21, 1953. Fourteen months before he had noted the onset of abdominal swelling and subsequently edema of the lower extremities and dyspnea.

He had massive edema of the lower extremities, ascites, an enlarged liver which extended 7 or 8 centimeters below the right costal margin, and a quiet heart. Blood pressure was 110/90, and the venous pressure was 24 centimeters. Total proteins were 4.4 grams per cent, the albumin fraction being 3.07. Fluoroscopic, roentgenographic and electrocardiographic studies were typical of chronic constrictive pericarditis.

He was operated upon on May 25. The pericardium was everywhere thick and adherent. As soon as it was split in the midline and dissection was begun to free it from the heart, the blood pressure rose from 100/60 to 150/100. The pericardium was removed on the right side back far enough to free the atrium and venae cavae, and on the left side beyond the level of the phrenic nerve. The excised tissue showed evidence of nonspecific chronic pericarditis.

Convalescence was uneventful. When he left the hospital on the ninth postoperative day he weighed 33 pounds less than before operation and was free of edema.

He has remained well; is working, and leading a normally active life. In December 1953 he was free of edema and weighed 45 pounds more than he had when he had left the hospital. Blood pressure was 142/78 and the venous pressure was 19 centimeters.

DISCUSSION

Of the 9 patients treated, 7 were classical examples of generalized chronic constrictive pericarditis. Five of the 7 are well for all practical purposes following operative pericardial resection. One who is remarkably improved has known rheumatic heart disease with mitral stenosis and insufficiency and his few residual

difficulties are thought likely to be the result of his valvular disease. One also is much improved although not well. Since she remains relatively free of symptoms in spite of failure to follow medical advice and her occasional difficulties are easily resolved by salt restriction, reoperation has not been advised. Since she has cardiac enlargement and active cardiac pulsations it is entirely possible that her residual symptoms are due to intrinsic heart disease rather than to continued pericardial restriction.

One of the patients had acute and chronic tuberculous pericarditis. Operative treatment was deferred in the vain hope that the process would become quiescent and had to be performed eventually with the patient in extremis. He did not survive. Another patient with severe hypertensive cardiovascular disease had no generalized pericardial constriction but adherent pericarditis associated with a nonresectable calcific stenosis of the superior vena caval ostium.

It seems evident that the first cardinal principle in the management of chronic constrictive pericarditis is early recognition of the condition and early operative treatment. The problem of diagnosis should not ordinarily prove difficult if this disorder is kept continually in mind as a possible cause of cardiac failure and particularly in those instances in which common causes of myocardial failure are not present. The importance of quiet heart sounds, poor cardiac pulsations as observed fluoroscopically or by kymographic study, elevated venous pressure, low arterial pressure and small pulse pressure, and low voltage and flat or inverted T waves in the electrocardiogram have been emphasized repeatedly in the literature. The demonstration by cardiac catheterization of elevated venous and right atrial pressures and a mid-diastolic ventricular plateau with or without right ventricular and pulmonary hypertension is very suggestive evidence, although it is becoming increasingly apparent that these findings are not pathognomonic, but may be observed in other conditions such as heart failure from other cause.

Occasionally a difficult diagnostic problem may arise. I have through error operated upon 1 patient who proved to have a normal pericardium. She was a 52 year old woman with exertional dyspnea, ascites, hepatomegaly, and edema of the lower extremities. There was no murmur. The venous pressure was elevated. Electrocardiograms showed low voltage in the QRS complex and ST-T alterations. Fluoroscopic and roentgenographic examination, although not typical of constrictive pericarditis, was compatible with this diagnosis. The heart was enlarged. Pulsations were reduced. Cardiac catheterization revealed a mid-diastolic right ventricular plateau without ventricular systolic hypertension or pulmonary hypertension. Radial artery tracings showed the pressure to be low and pulse pressure small (93 to 90/83 to 79). The precise cause of the patient's difficulty has not been clarified. Venograms revealed no block in the inferior vena cava. No improvement followed thyroid medication. Although at first unsuccessful, a rigid cardiac regimen ultimately has kept the patient in fair health. Diagnostic problems as difficult as this are rarely encountered and most cases of chronic constrictive pericarditis should be recognized with ease.

In cases of chronic constrictive pericarditis without evidence of acute inflam-

mation there seems little question but that operation should be advised once the diagnosis is established. Operation should be deferred only until the patient is in optimal condition and as free as is possible of water retention. In many patients, as was true in some of ours, bedrest, restriction of sodium intake and use of mercurial diuretics prove quite effectual. In some it may be advisable, in addition, to utilize carboresins. The value of digitalis seems unproved in general unless specifically indicated by some complication such as rapid auricular fibrillation. There are cases, however, in which such a regimen is without any apparent benefit. The patient in case 8, for example, had been treated intensively for a long time with rest, low sodium diet, diuretics and digitalis without effect. He had massive ascites, massive bilateral pleural effusion and massive edema of the trunk and all four extremities. Following operative resection of the pericardium he lost 63 pounds of weight within a period of three weeks and was free of edema, ascites and pleural effusion. In patients so resistant it is an obvious error to defer operation in the vain hope that continued medical management eventually will prove of help.

Up until recent years it has been agreed generally that one should make every effort to defer operative treatment in cases of active tuberculous pericarditis until evidence of activity ceased. This policy was based upon the relatively high incidence of persistence or spread of tuberculosis, sometimes with fatal outcome. On the other hand, watchful waiting in the presence of persistent and increasing cardiac constriction may end disastrously as it did in the patient in case 2. This patient well might have withstood operative decortication at the time of his admission. Because of active pericarditis, however, operation was put off until it finally had to be done with the patient in a near-terminal state. Holman recently has demonstrated that operation now can be undertaken in such patients with reasonable safety with the use of streptomycin.[6] He points out that all such patients should be treated first with bedrest, a nutritious diet, and antibiotics and that operation should be deferred until improvement is evident from reduction of fever and tachycardia and decrease in sedimentation rate, provided the effects of cardiac compression are not prominent, incapacitating, or progressive. If, however, the cardiac compression causes prominent, incapacitating or progressive effects he believes that operation should not be delayed. Such a policy would seem wise. In circumstances of this sort every effort obviously should be made to isolate and culture the tubercle bacillus if possible from pericardial fluid, sputum or gastric washings, to determine its sensitivity to the various antituberculous drugs available and to utilize those most effective preoperatively and postoperatively.

Lack of cessation of symptoms and signs after pericardial resection must be assumed to be due to inadequate decortication unless there is good evidence of the presence of intrinsic myocardial or valvular disease. In such cases cardiac catheterization may be of great help in planning the secondary operation. If, for example, right ventricular and pulmonary hypertension are demonstrated the objective should include adequate freeing of the left side of the heart. Occasionally failure to achieve a perfect result may be due to known valvular or

myocardial disease as is clearly the case in one of our patients (case 6) and possibly in another (case 3).

The choice of operative approach and the objectives of operative treatment are necessarily linked with the following questions: Compression of what part of the heart is most important in producing the signs and symptoms of chronic pericardial constriction? And its corollary, decompression of what part of the heart is most important in bringing about relief of these signs and symptoms? As Holman and Willet[7] have pointed out in their excellent review, it commonly has been thought by surgeons that it was hazardous and unimportant to free the auricles and the venae cavae. Some have believed that removal of the pericardium from the left ventricular area was most important while others have thought it was most important to free the right ventricle. Some have held the opinion that the decortication should be as complete as possible and that auricles, ventricles, and venae cavae should be freed.

There can be little doubt from a survey of the literature that the pericardial resection should be extensive. Numerous cases where a small resection has not brought relief and further decortication has been necessary have been reported. In instances in which the original pericardiectomy has not been successful, secondary resection of pericardium from over right and left ventricles has brought about a satisfactory result. For example, in 2 of Heuer and Stewart's patients the original operation apparently failed to free sufficiently the right side of the heart and secondary decortication of this area brought about relief of symptoms.[4] On the other hand, subsequent more extensive liberation of the left ventricular area was required in 2 of Neuhof's patients[3, 5] and in several reported by White, Alexander, Churchill and Sweet.[14] The latter authors pointed out that in 2 patients in whom the original pericardiectomy failed to bring about satisfactory improvement, cardiac catheterization revealed elevated pulmonary artery pressure. It is of interest that my only patient subjected to a secondary operation had right ventricular hypertension; that cardiac catheterization following the first procedure showed the vena caval and right atrial pressures to have been reduced somewhat; but the right ventricular hypertension to have persisted, and that subsequent removal of the residual pericardium overlying the left posterolateral border of the heart resulted in a cure. Holman and Willett mentioned personal cases in which they thought that failure would have resulted had not localized constrictions of superior and inferior venae cavae been removed. One of our patients without the usual signs and symptoms of constrictive pericarditis but with hypertensive cardiovascular disease with heart failure and the syndrome of superior vena caval obstruction had an adherent but apparently nonconstrictive pericarditis with an associated clacific stenosis of the superior vena caval ostium. Such clinical observations would make it evident that inadequate decortication of either the left or right sides of the heart may in certain instances result in failure.

Considerable light has been shed upon the problem by experiments in which localized constriction has been induced. Both Parsons and Holman[12] and Isaacs, Carter and Haller[8] produced ascites by localized constrictive pericarditis over

the right auricle and ventricle and they both failed to produce symptoms with constriction of the right auricle alone. The former produced ascites by partial ligation of the inferior vena cava and the latter by localized constrictive pericarditis over the left auricle and ventricle. The results of the study of Isaacs and his associates are very illuminating. With chronic constriction of the right side of the heart there developed systemic venous hypertension, hepatomegaly, ascites, peripheral edema, hydrothorax and decreased cardiac output. With left ventricular constriction there developed pulmonary hypertension and reduced cardiac output. Generalized constriction of the heart resulted in pulmonary hypertension, systemic venous hypertension, hepatomegaly, ascites, peripheral edema, hydrothorax and reduced cardiac output. They concluded that constriction of either or both ventricles is of primary importance; that constriction of auricles rarely if ever is of primary importance and that, although total excision of the pericardial scar is desirable, liberation of both ventricles is essential.

From the recorded clinical and experimental data it seems clear that the objective of surgical treatment should be as complete a removal of the diseased pericardium as is possible and that under any circumstance the ventricles must be freed. Although removal of the pericardium from over both ventricles would seem clearly of primary importance in the majority of cases, I believe, along with Beck and Holman, that an effort should be made to free the atria and great vessels as well. To be sure, it is highly unlikely that a case will be encountered in which atrial compression will result in such reduction in size of the atrium as to cause impedance of blood flow through it. On the other hand only by freeing the atria as completely as possible will it be possible to detect any constricting bands about the vessels emptying into it and not only can ascites be produced by partial ligation of the vena cava[12] but cases have been recorded in which the operator felt that such constriction existed and required release. In patients with right ventricular and pulmonary hypertension the operator must be particularly careful to free the left side of the heart as widely as is possible. It seems evident that the best results will obtain if the operator makes every effort to carry out as extensive a pericardial resection as can be accomplished safely.

A variety of surgical approaches have been utilized. Most of the earlier operations were performed through a left anterior extra pleural incision. A number of costal cartilages were removed, often the third, fourth, fifth and sixth. Sometimes a portion of the attached ribs was removed as well, and some surgeons also removed the left half of the sternum. There seems to be general agreement that such an approach does not provide the desired exposure. Beck and Griswold[1] suggested a bilateral parasternal approach through an H shaped incision. A medium stenotomy from the xiphoid to the second interspace has been used by Lilienthal,[10] Roberts and Wilson[13] and Holman and Willett.[7] Essentially this approach was used in my first case and the exposure was good. I have, however, come to prefer the complete sternal-splitting incision. Blalock,[2] who used this approach for sometime, now prefers a left anterolateral fourth intercostal transpleural approach. Recently, Muller[11] and Johnson and Kirby[9] have employed a

transsternal incision opened into both pleural cavities. Undoubtedly a number of incisions can be utilized effectively. Up to the present I have been impressed with the generally good exposure obtained through a complete sternal splitting incision with wide retraction of the divided edges of the sternum.

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A STUDY OF ONE HUNDRED AND SIXTEEN ANEURYSMS OF THE AORTA AND ILIAC ARTERIES WITH REMARKS CONCERNING SURGICAL ATTITUDES

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Syphilitic vascular lesions with surgical significance have been of considerable interest for years. More recently, a general awareness of the vascular problems posed by the lengthening span of life has occupied the attention of both the internist and surgeon. While congenital and traumatic aneurysms are seen occasionally in civilian practice, those aneurysms secondary to syphilis and arteriosclerosis are by far the most common lesions encountered.

An accumulating experience indicates that not only has the incidence of aneurysms increased, but a reversal of the ratios of syphilitic to arteriosclerotic aneurysms has occurred as well. The increased incidence and this reversal of ratio is well exemplified by the following reports.

Osler[12] in 1905 reported an incidence of 1 per cent and a ratio of 10 syphilitic aneurysms to 1 arteriosclerotic aneurysm. Kampmeier[7, 8] reported a similar occurrence and a ratio of 8 to 1. Lucké[10] found an occurrence rate of 2.3 per cent. Epstein[5] and Maniglia[11] reported a similar incidence. Both authors revealed an increasing incidence of arteriosclerotic aneurysms. The latter author found arteriosclerotic aneurysms to exceed those of syphilitic origin in the ratio of 1.0 to 0.4.

Because cardiovascular surgery presently holds forth a great variety of potentially satisfactory approaches to this problem, it seemed wise to analyze our experience at the King County Hospital in order to determine the proper management of our own hospital population.

CASE MATERIAL

During a six year period from 1946 to 1952, 3,313 autopsies were performed at the King County Hospital. An autopsy diagnosis of aneurysm was made in 116 instances (fig. 1), an over-all occurrence rate of 3.5 per cent. Both aneurysms of arteriosclerotic and syphilitic origin were included, but only aneurysms of the aorta and iliac arteries were recorded. Traumatic and dissecting aneurysms were not included. There were 13 patients with multiple aneurysms who had a total of 24 aneurysms. However, only the most significant lesion in each of these patients was recorded in this study. The majority, 81 aneurysms, was found in the last three years. It should be noted that there has been a progressive increase in the number of autopsies performed, but this explains only in part the marked rise in the number found.

In about one fourth of the cases the gross report of the prosector served as

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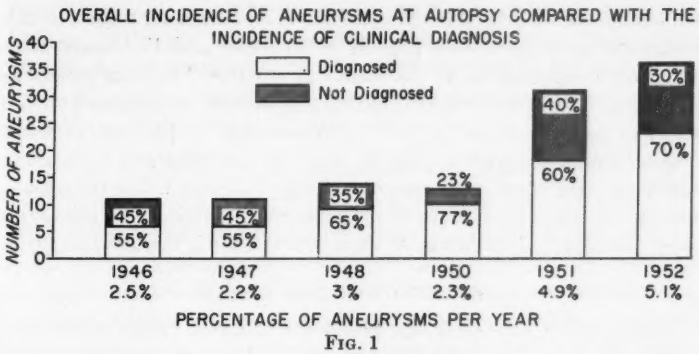

FIG. 1

the basis for classification of the anerusysm. Microscopy was available in the remainder, and in all when the etiology had been in question.

Arteriosclerotic aneurysms were present in 66 patients while syphilis was the cause in 50, a ratio of 1.3 to 1. This substantiates the reported increase of arteriosclerotic aneurysms already noted. Aneurysms were abdominal in 61 instances and thoracic in 55. Thoracoabdominal aneurysms were present in 6 patients. The apparent increase of arteriosclerotic aneurysms is both relative and absolute. The increase in the number of arteriosclerotic lesions probably is related to the greater number of aged people, while there has been a steady decrease in late and latent syphilis.[6]

AGE, SEX, AND THE RELATIONSHIP OF ETIOLOGY TO LOCATION

The average age of the 116 patients with aneurysms was 69.9 years. The youngest patient was 40 years, the oldest 89 years. The arteriosclerotic group averaged 70.9 years and the syphilitic group 68.8 years. These average ages are a reflection of the elderly patients cared for in this institution.

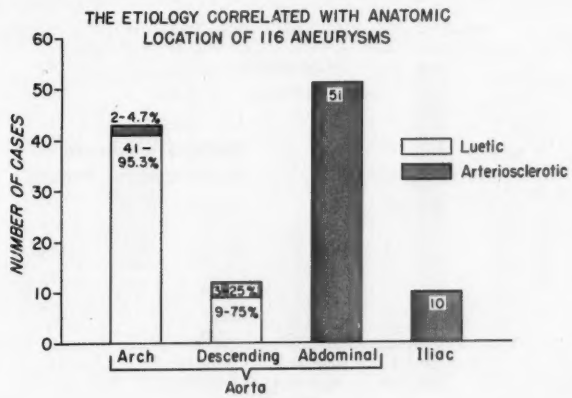

FIG. 2

There were 87 males and 29 females, a ratio of 3 to 1 among these 116 patients. Arteriosclerotic aneurysms were present in 55 males and 17 females while the aneurysms were syphilitic in 32 males and 12 females. This may reflect the decreased tendency for arteriosclerotic changes in females as compared with males.

Ninety-five per cent of aortic arch aneurysms and 75 per cent of aneurysms of the descending aorta were syphilitic. Only five intrathoracic aneurysms were arteriosclerotic and these were microscopically confirmed. By contrast, all 61 intra-abdominal aneurysms were of arteriosclerotic origin. The etiology correlated with the anatomic location of these aneurysms is depicted in figure 2.

ASSOCIATED VASCULAR DISEASE

At the inception of this study, the possibility of direct attack upon aneurysms by excision and suture and/or graft replacement had been seriously considered. Therefore, careful attention was given to the condition of the entire cardiovascular tree, especially that portion of the vessel immediately proximal and distal to the aneurysm which would be involved if graft replacement were considered.

The classification of sclerosis was quite simple. It was considered minimal if only subintimal plaques were present, but with a strong and pliable arterial wall; moderate when the aortic wall was less resilient than normal, but associated with prominent endarterial disease; and severe when the arterial wall was stiff and rigid with marked endarterial change.

While arteriosclerotic aneurysm was considered to represent a localized expression of a generalized arterial disease, the severity of the process found both proximal and distal to the aneurysm was greater than anticipated. Actually 80 per cent of aortas associated with arteriosclerotic aneurysms exhibited severe degenerative changes while 100 per cent exhibited at least moderate to severe changes. As a corollary, the greatest number of ruptures occurred in that group of patients with severe sclerotic changes (42 per cent).

The presence of severe aortic sclerosis in 27 of 50 syphilitic aneurysm patients

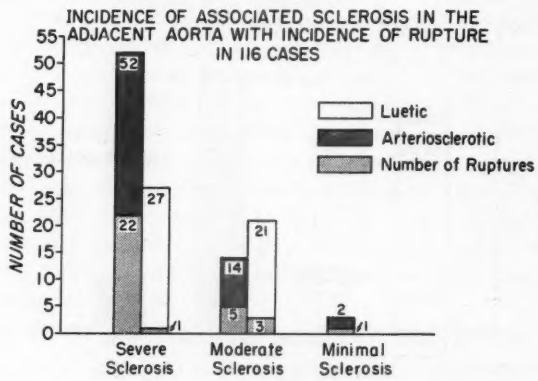

FIG. 3

(54 per cent) was quite surprising, and, in truth, the presence of moderate to severe changes in 96 per cent of patients with syphilitic aneurysms approximated that of the arteriosclerotic variety. Severe degrees of arteriosclerosis were present with all multiple aneurysms. Nine occurred in the arteriosclerotic group and four in the syphilitic group (fig. 3).

RELATIONSHIP OF CONTOUR TO RUPTURE

Each aneurysm was classified as being saccular, fusiform, and diffuse or cylindrical. The basis for this classification was as follows: Saccular aneurysms consisted of the aneurysms in which aortic openings generally were circular. Fusiform aneurysms had oval stomas, and the lateral wall of each was limited to one side of the aorta. Diffuse aneurysms were segmental arterial dilatations involving the entire circumference of the aorta. Tortuous aortas and nonlocalized dilatations were not considered to be aneurysms.

The relative frequency of the types encountered, with the incidence of rupture in each category is shown in figure 4.

The greatest number of ruptures occurred among the saccular aneurysms. There were 50 saccular aneurysms and 22, or 44 per cent, of these ruptured. There were 35 fusiform aneurysms and 10, or 29 per cent, of these ruptured. There were no fatal ruptures among the diffuse aneurysms. While certain formulas pertaining to the relationship of velocity and lateral pressure through inelastic tubes may represent valid explanations for the propensity of saccular aneurysms to rupture, it is apparent that in such instances the aneurysm increases in size at the expense of a limited area of arterial wall. Consequently, excessive thinning of a localized portion of the wall would tend to produce rupture more readily in contrast to aneurysms which extend over a greater area. It is of interest that the presence of a mural laminated thrombus did not appear to prevent rupture. This finding is in keeping with the observations of others.[1, 9]

SIGNS, SYMPTOMS AND DIAGNOSIS

The symptomatology referable to aneurysm is dependent upon many factors such as etiology, location, size and activity.

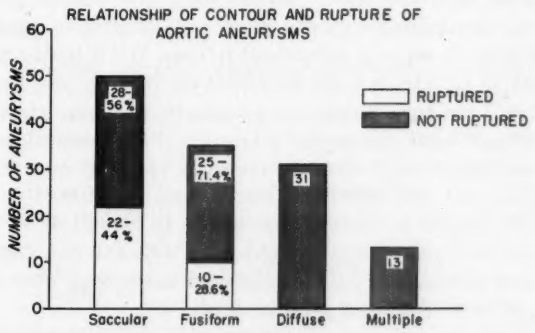

FIG. 4

PRIMARY SYMPTOMS AND SIGNS OF ANEURYSMS – (72 CASES)

LUETIC-(24 CASES)			
Pain	12	Mass	8
Dyspnea	4	Mediastinal fullness	4
Dysphagia	6	Tracheal tug	7
Cough	9	Shock	5
Fullness	3	Tracheal shift	2
Hematemesis	2	Bruit	2
Hemoptysis	1	Absent radial pulse	1
Hoarseness	2	Venous distention	1
ARTERIOSCLEROTIC-(48 CASES)			
Pain	34	Mass	22
Radiating pain	21	Shock	16 early
Vomiting	10	Tenderness	8
Urinary retention	1	Ileus	13
Weakness	3	Dullness	8
Melena	2	Bloody Thoracentesis	1
Hematemesis	1	Bruit	1

FIG. 5

Intrathoracic Aneurysms. Intrathoracic aneurysms primarily are syphilitic (50 of 55 cases). All syphilitic aneurysms in this series occurred in the chest. Of the 55 aneurysms in this category, 24 were symptomatic. Pain, cough, dysphagia and dyspnea were the most common symptoms (fig. 5). A triad of symptoms frequently encountered in aneurysms of the aortic arch was that of cough, dyspnea, and orthopnea. It is difficult to be sure, however, whether the symptoms were due to the mass per se or to congestive heart failure secondary to aortic valvular insufficiency. Associated aortic insufficiency was present in a sizeable number of these patients. Asymptomatic aneurysms seldom were clinically diagnosed. When this occurred, the physical signs of tracheal deviation or tug with a pulsatile mass in the thoracic or supraclavicular area were most often present; or the signs of aortic insufficiency indicated a need for fluoroscopy or roentgenologic examination. Roentgenologic examination was responsible for 24 diagnoses. The diagnosis was early in eight instances. Associated radiographic findings included vertebral erosion, an altered mediastinal shadow and, rarely, calcification in the aneurysm wall.

Aneurysms of the descending aorta often were large and manifested themselves by dysphagia, a sense of substernal fullness and a boring pain.

When rupture of an intrathoracic aneurysm occurred, it was usually massive. This occurred in 7 patients. Shock was present in all. Pain, when present, was severe and confused with myocardial infarction. Pain lessened as shock deepened. The severe progressive dyspnea observed probably was related to continued exsanguination and increasing intrapleural pressure. One may assume that progressive dyspnea is related more closely to blood loss as this symptom was present, also, in 3 other nonautopsied cases with external hemorrhage.

Intraabdominal Aneurysms. All 61 abdominal aneurysms were of arteriosclerotic etiology; 48 were diagnosed prior to death.

The patient with an asymptomatic aneurysm occasionally complained of an

increased abdominal girth or noted a pulsatile mass. The most consistent sign was the presence of a mass, frequently pulsatile, located in the epigastrium more frequently to the left of the midline and at times tender to palpation. The initial diagnosis was made by roentgenogram in 4 cases and was confirmatory in 14 others. The most frequent findings were the observation of a thin rim of calcium in the upper abdomen and a vague area of density with visceral displacement. Aortograms seldom were made.

When symptoms were present, pain was the most prominent complaint. The pain sometimes was poorly localized, but usually periumbilical or epigastric and had a boring quality. This symptom appears to be related to peritoneal stretching due either to aneurysmal enlargement or retroperitoneal hemorrhage. A tender mass was noted by 4 patients. A history of a vague sensation of epigastric fullness and soreness was elicited from several others. In some, the mass was tender to palpation. Pain was present in 34 of 48 symptomatic aneurysms, and in 24, it heralded a rupture. It is of considerable interest that some patients had experienced pain, not too severe, several days and sometimes weeks prior to death. Furthermore, in eight instances, small ruptures had sealed prior to the final lethal rupture. This sequence of events invalidates the impression that all ruptures are immediately catastrophic. The time interval between onset of pain and death due to rupture was adequate in many instances to allow time for a direct surgical attack. In short, pain in itself indicates an impending rupture, and, in fact, it may indicate a *leak*. However, even this does not necessarily mean immediate death.

Fatal aneurysmal rupture was always spectacular. Severe pain was present in 24 of the 27 ruptured aneurysms. (The remaining 3 patients died during the night unattended; consequently, no information was available.) The radiation of pain associated with rupture is indicated in figure 6. The radiation of pain frequently clouded the clinical picture. Seven patients were explored surgically with an erroneous diagnosis. Exploration was contemplated in 5 others; however, the terminal state of the patient tempered this decision. The severity of pain appeared to parallel the blood pressure; as the pressure fell, the pain diminished. The color of the skin was ghostly pale, but most patients were mentally alert in spite of considerable analgesic medication.

On abdominal examination, spasm and tenderness, most severe over the af-

RUPTURED ARTERIOSCLEROTIC ANEURYSMS – (27 CASES)

Pain associated with rupture	24	Palpable Mass		17
Radiation of pain	21	Increased size	4	
Back	6	Pulsated	5	
Flank	5	Decreased	1	
Hip – Leg	5	Surgery during rupture	7 cases	
Groin	2	Preoperative Diagnosis	Urinary retention	1
Chest	1		Volvulus	1
Shoulder	2		Perforated ulcer	4
			Pancreatitis	1

FIG. 6

fected area, were present. This was most prominent in those patients with retroperitoneal hemorrhage. Paralytic ileus was present early in 13 instances and in all but 1 of the 20 patients with retroperitoneal rupture. This combination of events frequently made palpation impossible; however, it was occasionally possible to palpate a mass. A femoral pulse was present in about 50 per cent of the patients. Rectal and pelvic examinations revealed tenderness or a tender mass mainly in patients with a ruptured iliac anerusym.

In the entire series, 72 (62 per cent) of 116 patients were diagnosed prior to death. Concomitant with the increased incidence of aneurysms in this institution, a greater percentage of aneurysms coming to autopsy are being diagnosed clinically. However, it is important to point out that too frequently the diagnosis still is made as a terminal event. A failure to think of aneurysm and the lack of familiarity with the symptoms and signs of aneurysm were responsible for several erroneous diagnoses. In general, abdominal aortic aneurysms can be diagnosed frequently by obtaining a good history and doing an adequate physical examination and the procurement of appropriate roentgenograms.

THE LIFE EXPECTANCY OF THE PATIENT WITH ANEURYSM AFTER THE DIAGNOSIS HAS BEEN ESTABLISHED

All patients, with one exception, regardless of the etiology or site of aneurysm, were dead within 37 months after the establishment of the diagnosis. The average survival period was nine months for the entire series. One year after diagnosis, 83 per cent of the patients had died. The patients with syphilitic aneurysms had an average survival period almost two and a half times greater than individuals with arteriosclerotic aneurysms (fig. 7). The high incidence of death in the first month following establishment of the diagnosis is in part due to the number of patients with apparent rupture at the time of emergency admission.

Of interest is the fact that of the total number of 24 patients with syphilitic aneurysm in whom the diagnosis was established clinically, only 3 (13 per cent) died of rupture of the aneurysm. By contrast, 20 (42 per cent) of 48 patients

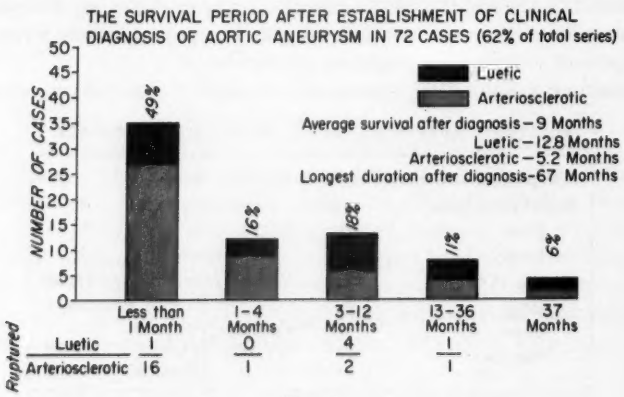

FIG. 7

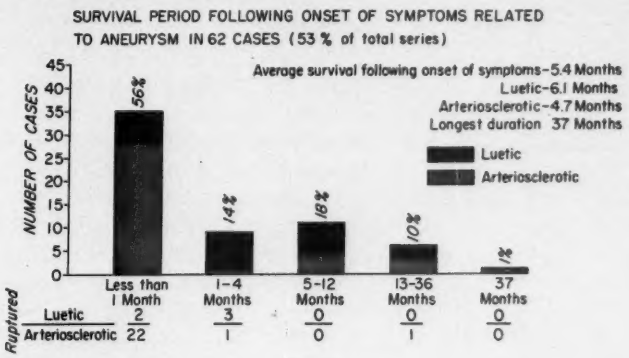

FIG. 8

with clinically diagnosed arteriosclerotic aneurysms died of rupture. It appears that the danger of rupture of the arteriosclerotic aneurysms is three times that of the syphilitic. Therefore, of all 72 patients diagnosed clinically (regardless of etiology), 23 died of ruptured aneurysms and 3 of *pressure* (36 per cent), and 46 (64 per cent) died of causes other than aneurysm. These facts regarding this hospital population raise two important questions, viz., which patient should be offered therapy? Should it be palliative or *curative*?

THE LIFE EXPECTANCY OF THE PATIENT WITH ANEURYSM AFTER SYMPTOMS ARE PRESENT

As might be expected, when symptoms referable to aneurysm are present, the patient's life is in jeopardy. The average survival for the 62 patients with symptoms was 5.4 months (fig. 8). The average difference in survival between symptomatic syphilitic and arteriosclerotic aneurysms was not striking. Of 25 patients with symptomatic syphilitic aneurysms, 5 (20 per cent) died of rupture, and an additional 3 (12 per cent) died of *pressure*. Therefore, 8 (32 per cent) of 25 patients with symptomatic syphilitic aneurysms died of their aneurysms. It should be pointed out that most syphilitic aneurysms were of the ascending aorta and frequently associated with aortic insufficiency.

The symptomatic arteriosclerotic aneurysm had by far the poorest prognosis. In all, 24 (65 per cent) patients with symptomatic arteriosclerotic aneurysm died of rupture. Twenty-two of these died within the first month after the onset of symptoms. Many arteriosclerotic aneurysms tabulated in the graph (fig. 8) were diagnosed because of the presence of symptoms. Consequently, it is not surprising that the mortality rate from ruptures shown in these two graphs is quite similar. More frequently than not, the abdominal aneurysm was diagnosed because of symptoms referable to it, and, less frequently, it was discovered as an incidental finding. Therefore, symptomatology referable to the aneurysm frequently coincides with the time of diagnosis. In any event, when symptoms are present, it usually means that rupture is occurring or is about to occur. This constitutes a firm indication for surgical therapy.

DISCUSSION

It is apparent that an aneurysm, be it syphilitic or arteriosclerotic, represents a local expression of a generalized disease. In no sense is the basic disease process altered by therapy of the aneurysm per se. The importance of this fact is made clear by the data presented. In the entire series of 116 cases of aneurysm, 35 patients died of the aneurysm (32 due to rupture, 3 due to *pressure*). Therefore, 30 per cent in this series died of causes directly related to the aneurysm, while 81 patients (70 per cent) died of other causes. Most died from complications secondary to their basic underlying disease or conditions associated with the aging process. Of 72 cases of aneurysms diagnosed clinically, 29 patients (40 per cent) died as a result of the aneurysms. Consequently, the remaining 43 patients (60 per cent) died of causes other than aneurysm within 37 months from the establishment of the diagnosis. Only 1 patient survived beyond this period. He died at 67 months but not of aneurysm. Furthermore, it should be noted that symptomatic aneurysm has a more serious prognosis. Of 62 patients with symptomatic aneurysm, 32 (52 per cent) died as a result of the aneurysm. Consequently, 30 patients, or 48 per cent, died within 37 months of other causes.

The prognosis of aneurysm appears to be closely related to the etiologic agent. Of the 66 arteriosclerotic aneurysms, 27 (41 per cent) ruptured fatally. Of the remaining 39 patients, 26 died of causes directly related to the basic disease process (heart failure, coronary occlusion, renal and cerebrovascular accidents). Consequently, in those patients with arteriosclerotic aneurysms, an almost equal number of patients are dying of their basic disease as are dying of the ruptured aneurysm.

Of the 50 syphilitic patients with aneurysms, only 8 (5 rupture, 3 *pressure*) (16 per cent) died as a direct result of aneurysm. Therefore, the risk of a syphilitic aneurysm is less than the risk associated with arteriosclerotic aneurysm. Granted that when rupture occurs, it is a spectacular event, the risk of rupture of the syphilitic aneurysm has been overemphasized when compared to the life expectancy of the generalized disease per se. Of the remaining 42 cases, 30 patients died as a direct result of syphilis (heart failure, coronary occlusion, cerebrovascular accident).

Consequently, the practical problem is to differentiate those who will die of aneurysm and should be treated, while avoiding those who will die for other reasons. It is apparent when one concerns oneself with the entire series that not all cases were diagnosed. The poor prognosis of aneurysm, after the diagnosis is established and when symptomatic, necessitates considerable judgment, not only in the decision to operate, but also in the method to be utilized. While it is apparent that one may guess wrong only once, it is believed that a reasonably correct appraisal of the situation on both scores can be made on clinical grounds alone.

In arriving at a critique for operative management of the patient with aneurysm, the advantages and disadvantages of each form of therapy must be thoughtfully considered in terms of the patient's prognosis if left untreated. Present methods which appear to have merit can be considered, for purposes of discus-

sion, under *palliative* and *curative* methods. The *palliative* methods consist of the utilization of the intravascular wiring technic, perfected by Blakemore,[3] and the application of fibrosing materials.[13, 14] The *curative* are concerned with excision of the aneurysm.[2, 4] *Palliative* methods, although wanting in certain respects, are utilizable in most cases of aneurysm, regardless of the etiology and the site. Excisional methods with lateral repair or excision of the aneurysm with a replacement by homologous graft might be considered a more direct approach to the problem, but, of course, must be considered *curative* in the most restricted sense. The latter methods appear to be restricted in their applicability, conditioned by many factors including the location of the aneurysm.

Ascending Aorta. Most aneurysms in this area are of syphilitic etiology and often associated with aortic insufficiency. The prognosis of the patient is more directly related to the heart failure secondary to aortic insufficiency than to the danger of aneurysmal rupture. Therefore, while the direct approach with excision and lateral reconstruction appears to have great merit, in our experience it does not appear to be feasible in many instances. The *palliative* measures here will be of definite use.

Arch of the Aorta. These aneurysms usually of syphilitic etiology may be approached by excision with lateral reconstruction or the utilization of either of the *palliative* measures. In most instances these aneurysms will not be associated with aortic insufficiency. Therefore the direct approach, where feasible, appears best. Undoubtedly, successful replacement of the aortic arch will be accomplished and reported. However, the indications for such a procedure undoubtedly will be few.

Descending Aorta. Aneurysms of the descending aorta distal to the left subclavian and extending to the renal arteries, while mainly of syphilitic origin, will be of both a syphilitic and arteriosclerotic origin. Here, again, both *curative* and *palliative* measures are available.

The *curative* methods of lateral reconstruction and excision with graft replacement may be indicated. If replacement by graft is considered, it should be accompanied by simultaneous shunting. Otherwise, damage to distal structures and to the spinal cord may occur. The number of intercostal arteries which may be sacrificed without spinal cord damage is not yet known. In certain instances there appear to be no limiting factors. This may be true in the young or in patients with aneurysms where gradual occlusion of the ostia may have occurred. However, in an elective situation one should be cognizant of our lack of knowledge concerning the accurate pattern and variation of the blood supply to the spinal cord via the intercostals. One necessarily would consider the patient's over-all prognosis quite carefully before a decision is made between *palliative* and *curative* methods.

Abdominal Aorta. Asymptomatic: Most aneurysms of the abdominal aorta occur below the renal arteries. The methods of therapy available in such cases are both *palliative* and *curative*. Either palliative method may be adequate in the prevention of rupture, often allowing the patient sufficient palliation to live out his lifespan with death as a result of other complications of arteriosclero-

sis. In certain instances, when the patient has few of the stigmas of severe generalized disease, excision with replacement by homologous graft seems apropos. It should be pointed out, however, that the vascular sclerosis in this series was extremely severe. One would have to balance carefully the risk of the operation versus the gain.

Symptomatic: Most symptoms of arteriosclerotic abdominal aneurysms, especially pain, indicate impending rupture or early leak. In this situation, in our opinion, based upon the data herein presented, only the *curative* methods are applicable, either by lateral reconstruction or excision with homologous graft replacement. The operation should be considered a life-saving approach. Granted that the risk may be high, there is everything to be gained and nothing to lose. The utilization of *palliative* principles in this situation is without any merit whatsoever.

The field of cardiovascular surgery is in an extremely dynamic phase. However, enough information is known about the methods discussed, that this outline as a working guide to the management of this particular hospital population seems reasonable. The method of excision with graft replacement offers a direct approach to the problem. While we have used homologous graft (freeze-dry) replacement for abdominal aneurysm, our reluctance to apply this principle widely is conditioned by the life expectancy of our patients with aneurysm seen in this hospital. The utilization of pure dicetyl phosphate powder rather than impregnated paper has been satisfactory to date in the prevention of rupture.

CONCLUSIONS

During the period from 1946 to 1952, 116 patients with aneurysm of the aorta and iliac arteries have been diagnosed at autopsy at the King County Hospital.

A study of this material indicates that the incidence of aneurysm has increased together with an improvement in the percentage of aneurysms being diagnosed clinically.

Arteriosclerotic aneurysms account for the increased incidence; the ratio of arteriosclerotic to syphilitic aneurysms was 1.3 to 1.

The prognosis of the patient with either a syphilitic or arteriosclerotic aneurysm is poor. However, the aneurysm per se is merely a localized expression of a serious generalized disease.

Of 72 patients diagnosed clinically, 29 (40 per cent) died as a result of aneurysm. The remaining 60 per cent died of causes other than aneurysm. All died (with one exception) within 37 months after diagnosis.

Of 62 patients exhibiting symptoms related to aneurysm, 32 (52 per cent) died of aneurysm. The remaining half died of other causes. Both groups died within 37 months of the onset of symptoms.

Of 66 patients with arteriosclerotic aneurysm, 27 (41 per cent) died of fatal rupture. An almost equal number (26 patients) died of causes related to the underlying basic disease.

Of 50 cases of syphilitic aneurysm, only 8 patients (16 per cent) died as a result of aneurysm; 30 of the remaining 42 died of complications directly related

to generalized syphilis. It appears that the arteriosclerotic aneurysm is more dangerous by far than the syphilitic aneurysm. The risk of aneurysmal rupture of the syphilitic type has been overemphasized in regard to the over-all prognosis of generalized syphilis.

The occurrence of moderate to severe generalized aortic and iliac arteriosclerosis is approximately the same regardless of the etiology of the aneurysm. The most severe forms, however, were noted in the arteriosclerotic group.

A discussion has been presented of our current thinking regarding the surgical approach to the problem based upon this study of our hospital population.

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PROGRESS IN STUDY OF STRANGULATION INTESTINAL OBSTRUCTION

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The resurge of interest in strangulated bowel obstruction warrants a re-evaluation of the subject in the light of current findings. Some recent experimental findings are summarized in the hope that further interest in the problem will be aroused.

It is well to define our terms at the outset. Dorland's dictionary defines strangulation as "an arrest of the circulation in a part, due to compression". Wangensteen has said that strangulating obstructions are "characterized by the presence of a block in the bowel with the added factor of infarction of the imprisoned segment of bowel".[24] Reinus has divided obstruction into three categories as follows: (1) simple obstruction, (2) obstruction with imminent strangulation, and (3) obstruction with strangulation. "Imminent strangulation is considered to mean that serious changes of a vascular nature had occurred in a segment of small intestine and that resuscitation of the bowel for varying periods was necessary at operation. . . . Obstruction with strangulation was considered to mean that hemorrhagic infarction had occurred."[20]

A research problem should have some clinical bearing and not merely be an interesting exercise in experimental work. Several recent analyses have shown that the incidence of intestinal obstruction still is very high and that the mortality rate from intestinal obstruction, while constantly decreasing, still is significant. Furthermore, the mortality rate from strangulation obstruction is not being reduced with the same rapidity as is the mortality rate from simple obstruction. Becker's analysis of the experience at Charity Hospital in New Orleans from 1940 to 1950 demonstrated the importance of this condition in our area.[2] During this 10 year period, 1007 patients with acute mechanical intestinal obstruction were admitted to the Charity Hospital. That is a 10 year average of 1 case admitted every three and a half days. Of the total number, 27.7 per cent were cases of strangulation obstruction. There was an over-all mortality rate of 18.7 per cent. However, the mortality rate in simple obstruction was 14.1 per cent, as contrasted with a mortality rate of 30.5 per cent in strangulation obstruction. While Becker's figures represent a definite improvement over the 65 per cent mortality rate reported by Miller[16] from the Charity Hospital in 1929, the present mortality rate for strangulation obstruction still is very high. In Becker's study of acute adhesive ileus there were 88 cases of strangulated obstruction and 324 cases of simple obstruction.[1] In simple obstruction the mortality rate was 6.7 per cent, whereas in strangulated obstruction there was a mortality rate of 30.6 per cent.

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"The absence or presence of signs of irritability of the parietal peritoneum suffice usually to differentiate" simple and strangulation obstruction, according to Wangensteen.[24] Becker's work on acute adhesive ileus would indicate that "the absence or presence of fever, tachycardia, leukocytosis, a palpable mass, and peritoneal irritation does not suffice to differentiate the two varieties of obstruction."[1] If the differential diagnosis between simple and strangulation obstruction cannot always be made and if the mortality rate of strangulation obstruction is so much higher than that of simple obstruction, then it becomes more important to discover the causes of death in strangulation obstruction and some therapy that can lower the mortality rate.

It is with these thoughts in mind that the present experimental work on strangulation obstruction has been done, and it is with the hope of finding some means of combatting what we believe to be the cause of death that the work is being continued.

Re-evaluation of experimental strangulation obstruction in the light of current knowledge regarding hemorrhage, shock, dehydration and electrolyte loss was necessary first.[17] A standard type of strangulation obstruction was used in dogs, in which an obstruction was created by dividing the bowel and closing the ends so that complete obstruction unquestionably would be obtained. The bowel was divided 100 to 150 cm. from the ligament of Treitz and a segment of the proximal portion strangulated by ligation of all the veins to a 30 cm. segment. Ligation of the veins alone was selected because Scott and Wangensteen had shown that this gave a shorter survival than interference with the arterial or the arterial and the venous blood flow.[21, 22, 23] Test conditions were made as severe as possible so that any prolongation of survival would be more significant. Rubber tubes in the peritoneal cavity and a plastic tube in the strangulated bowel permitted study of the various fluids during the postoperative period. The animals were observed constantly from operation until death and were treated with electrolyte solutions, blood, plasma, or gelatin, as indicated. The bowel content, peritoneal fluid, and blood were subjected to chemical and bacteriologic studies.

The animals survived from 28.25 to 48 hours following operation, with an average survival time of 36 hours. This was a prolongation of life over previous results, since untreated animals with a similar length of strangulated intestine had survived previously only 7 to 24 hours. At autopsy, the strangulated segment of intestine was black, necrotic, gangrenous, elongated and dilated, but was not perforated. A very sharp line of demarcation at the beginning and ending of the strangulated portion was noted. The peritoneum, while showing generalized areas of subserous hemorrhage and congestion, never gave the picture of a typical bacterial peritonitis.

The clinical condition of these animals was quite interesting. On recovery from anesthesia, they would walk about in their cages and would seem to be in good condition until one to four hours prior to death, when a very sudden change occurred. The animals became much sicker, their retching and vomiting became more severe, they began to have respiratory difficulties, and frequently in the period immediately before death there would be convulsive movements. Femoral

pulsations, palpable until several minutes before death, and observation of hemoglobin, hematocrit, and plasma protein all showed the animals did not die in shock.

In connection with the changes observed clinically, there was a constant sequence of events in the peritoneal fluid, bowel contents, and blood. In the early postoperative period, the peritoneal fluid was reddish, coagulable, odorless, and was not hemolysed. When the clinical condition changed abruptly, the peritoneal fluid changed to a blackish, foul-smelling noncoagulable and severely hemolysed fluid. This peritoneal fluid very closely resembled the fluid which had been noted in the strangulated segment much earlier. The nitrogenous components of the peritoneal fluid rose abruptly when the black fluid appeared. Very shortly after the appearance of this black fluid in the peritoneal cavity, a similar rise in the nitrogenous components of the blood was observed. Bacterial studies on the peritoneal fluid showed that it frequently was sterile 14 to 20 hours following operation, but after this the organisms which previously had been cultured from the bowel lumen began to appear in the peritoneal cavity and were present thereafter. Bacteremia was not found to play a significant role in any of the dogs.

Spectrophotometric examination of the bowel contents, peritoneal fluid and blood showed the colored pigment to be a derivative of hemoglobin, although it was not identified. The new pigment substance was found in the lumen of the strangulated bowel very shortly after strangulation, but did not appear in the peritoneal fluid until it changed from red to black. In some animals in which the black peritoneal fluid was observed for relatively long periods, a similar changed pigment was observed in the blood stream. The spectrophotometric observations demonstrated that substances formed in a strangulated segment of bowel are only absorbed after they have passed through the bowel wall into the free peritoneal cavity. This refuted the many previous suggestions about alternate routes of absorption from a strangulated segment.

Since the black peritoneal fluid was so closely correlated with the death of the animal, the presence of some lethal factor in this changed fluid was evaluated by injection experiments.[18] Peritoneal fluid was injected into normal dogs, in quantities insufficient to cause any disturbance of the circulatory system of the recipient animal. The early or red peritoneal fluid had no effect upon the recipient animal, while the black or dark, or late peritoneal fluid was injected with lethal results. Therefore, this late or dark peritoneal fluid contained some lethal factor.

Histologic study of the strangulated bowel focused our attention on the possible role of the gas gangrene organisms.[6] Review of the bacteriologic studies from the original series, showing only three organisms uniformly present in the peritoneal fluid—hemolytic clostridia, B. coli, and streptococci—strengthened the impression of the importance of the gas gangrene organisms. Accordingly, a technic was sought whereby we could study the gas gangrene organisms in the peritoneum of otherwise normal dogs. It was also essential to determine whether or not a picture somewhat similar to that of strangulation obstruction could be

obtained without its complicating factors such as nausea, vomiting and blood loss due to hemorrhage.

A completely isolated, closed, devascularized and separated loop of bowel placed in the peritoneum of an otherwise normal dog answered these requirements.[10] This technic was superior to strangulation obstruction in that: it avoided the vomiting due to obstruction, it prevented hemorrhage and fluid loss into the strangulated loop of bowel, it prevented absorption via the lymphatics since this was an isolated loop, and it prevented the interaction of bacteria, gastrointestinal secretions and blood in the lumen. To study the effects of autolysis of bowel tissues, autoclaved sterile bowel was placed in the peritoneum of normal dogs without effect. The importance of the gas gangrene organisms was then shown by placing sublethal doses of *Cl. welchii* with autoclaved sterile loops of bowel into the peritoneal cavity of normal dogs. The lethal results were due to the growth of the *Cl. welchii* in the presence of devitalized tissue. Since the sequence of events was similar to that in strangulation obstruction, the death of dogs which were otherwise adequately treated for strangulation obstruction was due to the effects of the *Cl. welchii* toxin. When the abnormal nature of autoclaved bowel tissue was questioned, bowel segments were sterilized by prolonged contact with the peritoneum. Fresh sterile bowel was prepared by allowing an isolated, well vascularized loop of bowel to remain open in the peritoneal cavity for prolonged periods, at the end of which time this loop could be cultured with negative results.[12, 13] Removal and injection of this loop with sublethal doses of *Cl. welchii* gave a fatal result, showing that bowel tissue sterilized by any means plus *Cl. welchii* will give lethal results.

Clinical confirmation of these experimental findings was sought by study of all patients with strangulation obstruction who were submitted to surgery in seven of the large Philadelphia hospitals over a two year period.[4] When any patient with suspected strangulation obstruction was submitted to surgery, the peritoneal fluid was subjected to both spectrophotometric and bacteriologic analyses. Correlations between clinical course, therapy, response to therapy, and laboratory findings with spectrophotometric and bacteriologic analyses of the peritoneal fluid were attempted. Of the 18 patients submitted for study, 11 were not examples of strangulation, and there was no change in the spectrophotometric observations in any of these patients. In 3 patients, strangulation was thought to be present, but was not thought to require resection. None of these had any changes in spectrophotometric observations. In 4 patients, strangulation was present and resection was essential. Of these 4 patients, only 1—and this was the only one in the entire series—showed any abnormality of the spectrophotometric curves. This one had changes which were quite similar to those observed experimentally.[11] The peritoneal fluid in cases of simple obstruction was essentially negative bacteriologically. In strangulation obstruction, positive bacteriologic findings in the peritoneal fluid were more common. Positive bacteriologic findings in the peritoneal fluid in any type of obstruction were more commonly associated with postoperative complications than were negative bacteriologic findings. The

infrequent clinical occurrence of positive changes in the spectrophotometric analysis of the peritoneal fluid served to de-emphasize the importance of this as a clinical finding, while in contrast, the more frequent occurrence of positive bacteriologic results in the peritoneal fluid showed a higher correlation, and therefore seemed to be of more importance both clinically and experimentally.

Biliary and pancreatic secretions have been emphasized by others as the major cause of death.[19] They attempted to reproduce the peculiar spectrophotometric curve by various combinations of biliary and pancreatic secretions, blood, and gastrointestinal secretions. To evaluate the importance of these secretions, we studied a special type of strangulation obstruction in which biliary and pancreatic secretions were eliminated from the strangulated, obstructed loop.[9] After recovery from a modified Mann-Williamson operation, dogs were subjected to reoperation and the creation of a strangulation obstruction above the point of re-entrance of the biliary and pancreatic secretions. A long interval between operations, and the complete obstruction obtained by dividing and closing the ends of the bowel insured the absence of both biliary and pancreatic secretions from the strangulated loop. Under these conditions, the dogs again failed to survive, showing that neither biliary nor pancreatic secretions, nor a combination of the two, was an essential component of the lethal result in strangulation obstruction.

Let us return to the specific role of bacteria. The advent of antibacterial agents which could render the bowel relatively sterile seemed to open a new approach to the study of strangulation obstruction. If the bowel could be freed of bacteria prior to operation, and the animal maintained postoperatively with the usual supportive measures plus adequate antibacterial protection, then it should be possible to obtain indefinite survival, if our original thesis was correct. One group of dogs was prepared preoperatively with oral sulfathaladine and streptomycin and parenteral penicillin. A second group of dogs was prepared with oral aureomycin and parenteral penicillin. Both received intramuscular penicillin and intravenous aureomycin postoperatively. There was no significant difference between the two groups of animals.[7, 8] These dogs survived from 28.5 to 116 hours with an over-all average survival of 71.5 hours. This was twice the survival time obtained in the animals that received no antibiotics. In several of the dogs which died, autopsy did not explain death satisfactorily and it was not possible at that time to explain why indefinite survival had not been obtained. The uniform clinical picture noted in the original series was not observed in this group. The sudden change in the peritoneal fluid did not occur. The death of some of these animals came suddenly and unexpectedly and was not explained on the basis of shock, electrolyte imbalance, dehydration or by any of the autopsy findings. In no case were the bacteriologic findings thought to be correlated with the cause of death. Of outstanding importance was the fact that histologic studies of the strangulated bowel showed a relatively normal appearance in contrast to the almost complete destruction in the nonantibiotic series. In addition, somewhat similar results had been obtained by Blain and Kennedy[3] using penicillin alone in the postoperative period. We were unable to explain our failure to improve upon their results.

Some time after our work was completed, Lepper and associates[14, 15] showed that excessive doses of aureomycin given intravenously could be fatal. Review of our data revealed that our animals had received intravenous aureomycin in the lethal range and some of the animals whose death could not be otherwise explained had histologic findings compatible with those described for aureomycin toxicity. This immediately suggested that further benefits might be obtained with preoperative bowel sterilization combined with postoperative antibiotics, provided a nontoxic route of administration could be found.

A nontoxic means of giving aureomycin has formed the basis of our most recent series of experiments.[5] It also has been the basis of the most gratifying results to date, since it would tend to show that the original contention regarding the importance of bacteria in the cause of death in strangulation obstruction is indeed a valid theory.

Within the past year we have conducted a series of experiments in which preoperative bowel sterilization has been utilized with postoperative intramuscular penicillin and aureomycin delivered directly into the strangulated segment through a plastic tube. To date, we have a series of 5 consecutive dogs subjected to this procedure with survivals being limited only by the time of reoperation. The animals survived from 118 to 142 hours with the average survival being over 120 hours. If adequate nutrition could be supplied by parenteral means, we believe that these dogs could withstand this type of strangulation obstruction indefinitely. However, nutritional losses became a major factor in a very short time and our inability to provide adequate parenteral nutrition has severely curtailed the interval between operations.

Observation beyond 116 hours does not seem valuable because the appearance of the bowel indicates that the period of maximum danger has passed and that the bowel is returning to a relatively normal state. Comparison of bowel from dogs which have survived a strangulation obstruction by virtue of antibiotic protection with bowel not protected by antibiotics shows a very marked difference in the two specimens on both serosal and mucosal sides. One of the most significant findings is the normal appearance of the mucosal surface. An even more striking finding is the relatively normal histologic appearance of the strangulated bowel, particularly in contrast with strangulated bowel from nonantibiotic experiments. In the antibiotic-protected animals there is hemorrhage and edema, but the mucosa is normal. In the animals which did not receive antibiotics destruction is complete from mucosa through serosa. Since the only essential difference between the two series is in the use of antibiotics, we cannot help but conclude that antibiotic protection is an extremely important feature in the cause of the fatal outcome in strangulation obstruction.

There are some other interesting observations in the antibiotic-protected animals. The rare episodes of hematemesis are in rather marked contrast to the frequent occurrence of hematemesis in all the other series. The peritoneal fluid in these animals never changed its appearance and did not contain clostridia. The bowel lumen, which was cultured in all experiments, did not contain clostridia.

Fluid balance studies have been calculated on a milliliter per kilogram per

24 hour basis, since this was the only means of reducing all values to a common denominator. The full significance of these results is not yet known. Both blood requirements and total intake in the latest series were significantly lower than in previous series. The lower blood requirements were not supplemented by additional gelatine or electrolyte intake. Output was also less. The decreased amount of vomitus in the latest series must be one of the more important features in the reduced fluid requirements. These observations on fluid balance have posed more questions than they have helped answer. Does the absence of bacteria in the bowel lumen at the time of strangulation have something to do with the amount of bleeding into the bowel lumen following strangulation? Do these two factors combined play a role in decreasing the amount of vomitus in the poststrangulation period? Why does bleeding not occur into the lumen of an antibiotic prepared strangulated bowel?

Work is continuing in an attempt to make some more practical clinical applications of the experimental observations. Even though it is impractical on a clinical basis to have an antibiotic prepared bowel in those patients who are going to have a strangulation obstruction, a thorough knowledge of the pathogenesis and the altered physiology which occurs in strangulation obstruction is essential before rational therapy can be outlined. It is in an attempt to find the pathogenesis of this condition that we are preparing animals in the manner we have outlined.

The pathology of *Cl. welchii* infections is being studied in an attempt to correlate the findings in experimental strangulation obstruction and fatal gas gangrene infections.

SUMMARY

Late in the course of experimental strangulation obstruction there are changes in the peritoneal fluid detected in bacteriologic content, chemical and spectrophotometric analyses, white blood cell counts, coagulability, hemolysis and odor.

Spectrophotometric examination of the bowel contents, peritoneal fluid and blood stream has shown a new and unidentified pigment which has served to establish the route of absorption in strangulation obstruction. This route is from the strangulated bowel across the bowel wall into the peritoneal fluid and then from the peritoneal fluid into the systemic circulation.

Injection studies have shown the late peritoneal fluid to be lethal, while the early unchanged peritoneal fluid has no effect.

The clinical course of patients with strangulation obstruction can be correlated more closely with the bacteriology of the peritoneal fluid than with the spectrophotometric studies of the peritoneal fluid.

Sterile tissues and sublethal doses of *Clostridium welchii*, although ineffective individually, in combination produced fatal results similar to those noted in strangulation obstruction, so that the fatality of strangulation obstruction was attributed to toxins of *Clostridium welchii*.

Proper attention to fluid, electrolyte and blood requirements significantly increased the survival of dogs with experimental strangulation obstruction.

The course of experimental strangulation obstruction was not significantly altered by the removal of activated pancreatic juice, duodenal or biliary secretions from the strangulated segment.

Preoperative bowel antisepsis combined with postoperative antibacterial agents significantly prolonged life over all previous technics.

Intravenous aureomycin in higher doses may be fatal to either animals or people.

Preoperative intestinal antisepsis combined with postoperative antibiotics, delivered directly into the strangulated segment, prolonged life of animals with experimental obstruction indefinitely.

The gross and histologic appearance of the strangulated segment of animals subjected to strangulation obstruction with antibiotic protection has shown only minor variations from the normal, indicating that permanent protection probably has been achieved.

Clinical applications of this experimental work have been suggested and are under current study.

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THE PROBLEM OF THE OVERLOOKED COMMON DUCT STONE

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THE PROBLEM

Most of the problems associated with the surgical management of biliary tract disease have been studied extensively during the past 50 years. There are remarkably few figures available, however, that indicate the incidence of stones in the common duct that have been overlooked at the time of cholecystectomy or choledochostomy. Accurate information on this point is difficult to obtain and even the closest scrutiny of any series of records will leave the investigator with some doubt as to the correctness of his findings. In table I, a few of the estimates appearing in the medical literature are listed. At the Buffalo General Hospital, approximately 1 patient in 4 with stones in the common duct that is explored leaves the operating table after exploration with one or more stones still in the duct. This fact became evident as a result of a survey of the operations performed on the common duct at this hospital during a nine year period which was made by Dr. W. S. Walls[17] and reported before the New York State Medical Association in May 1953.

The importance of this problem is indicated either explicitly or implicitly by almost everyone who has written about biliary tract disease and any surgeon of experience has only to refer to his own case records to emphasize the point. Thirty years ago, W. J. Mayo[11] noted that stones in the common duct were overlooked in one-third of the patients who died following choledochostomy with exploration. E. L. Young[18] in 1929 studied 67 autopsies of patients dying at the Massachusetts General Hospital following operations on the biliary tract and found that 27 patients (40 per cent) had had stones in the common duct overlooked at the time of operation. His rather whimsical conclusion was as follows: "Following operations on the biliary tract where calculi were present the percentage of failure in the removal of these stones was from 16.4 to 61.3, depending on the optimism or the viewpoint of the surgeon." Hicken[9] more recently has stated that 95 per cent of secondary operations on the biliary tract and 88 per cent of biliary fistulas are due to retained stones. At best, a stone remaining in the common duct after choledochostomy and exploration is embarrassing for the surgeon and disappointing for the patient. At worst, such a stone may be the direct or indirect cause of the patient's death.

The feeling of frustration which the speaker experienced some time ago upon noting an overlooked stone on a postoperative cholangiogram following what was thought to be a perfectly complete and satisfactory exploration of the com-

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TABLE I

Incidence of overlooked common duct stones

			Number of Cases
		%	
Bruning, A.[3]	1912	20	100
Hughes, C. R.[10]	1948		
First operation		27	46
Second operation		25	19
Hicken, N. F.[7]	1950	12	100
Glenn, F.[5]	1952	12	56

TABLE II

Indications for exploration of the common duct

(Colcock, B. P.—1948)[4]

1. Presence of jaundice.
2. Dilatation of the duct.
3. Abnormal thickness of the duct wall.
4. Presence of small stones in the gallbladder.
5. Palpation of a stone in the duct.
6. Presence of sludge in the duct.
7. Presence of pancreatitis.
8. Absence of stones in the gallbladder combined with a history of biliary colic.

mon duct, has prompted a more careful consideration of this problem and a study of the results of others in their attempts to deal with it.

It might be said that if all surgeons explored carefully and properly every common duct that they examined, fewer stones would be overlooked. It may be doubted, however, if the improvement would be very great. The indications for opening the common duct and the technic of its exploration are fairly well standardized (Table II). Colcock[4] in 1948 listed the indications for exploration. These are fairly uniformly observed throughout the country.

At our own hospital, exploration of the choledochus is done in a more or less routine fashion. Probes and forceps are passed up and down the duct and palpation, frequently from both sides of the operating table, is done carefully with and without a probe in place. After all detectable stones have been taken out, dilators increasing in size up to 5 or 6 millimeters in diameter are passed through the ampulla of Vater. The final maneuver usually consists of lavaging the duct system with saline solution injected through a small catheter which is inserted into the duodenum before its withdrawal. Despite all of this, stones both large and small continue consistently to escape detection. These stones usually are impacted in the lower end of the duct or lodged above within the substance of the liver where they cannot be palpated. Much variation in the consistency of the pancreas and the impression of lobulation which it gives to the exploring finger can be very confusing. Even though one or more fingers are inserted into the

avascular tissue plane posterior to the duodenum and head of the pancreas, one still can be misled.

POSSIBLE SOLUTION OF THE PROBLEM

If there is a problem of some magnitude and importance concerning the overlooked common duct stone, the obvious question is: What can be done about it? There would appear to be three viewpoints that might be worthy of thought. These are as follows:

1. Accept as inevitable that a sizeable percentage of patients will continue to have overlooked stones and attempt their surgical removal again as they become evident or sometime in the future if they become troublesome.

2. Attempt a course of treatment designed to cause fragmentation and dissolution of the stone and encourage the fragments to be spontaneously passed.

3. Attempt to decrease the incidence of the overlooked stone by the use of operative cholangiography.

1. Reoperation. It always has been our opinion that with other things being equal the known presence of a common duct stone was a sufficient indication for taking it out. Some of the best known surgeons in this country, however, have recommended that the unfortunate patient with an overlooked stone should be discharged from the hospital with no attempt being made to take it out unless trouble occured in the future. In our own clinic, about one fourth of the patients with overlooked stones are treated in this manner. This I believe, however, is not a good routine procedure. Of course, there well may be the occasional patient in whom the risk of a second operation would be of such magnitude as to more than outweigh the risk of leaving the stone alone and hoping for the best.

Common sense, however, would seem to indicate that no one would leave a stone in a duct by preference. The likelihood of a stone being present was sufficient indication for exploring the common duct with its increased mortality and morbidity rates in the first place. The known presence of a stone should be an equally strong indication for its removal no matter how embarrassed and reluctant the surgeon might be to explain the situation to the patient and impose upon him the risk and expense of another laparotomy. In the absence of controlled studies to indicate the advisability of one course of action over the other, the decision in this matter must rest on clinical judgment. In any event, I am sure that all will agree that any reasonable method by which the necessity of making such a decision may be avoided is very much worthwhile.

2. Dissolution and Fragmentation of the Stone. In 1939 Pribram[14] reported his remarkable success in dealing with overlooked common duct stones by the repeated injection of small quantities of ether and paraffin oil through the T tube in the immediate postoperative period. Again, in 1947 he reported complete success in treating 51 consecutive patients by this method.[15] In his opinion only those gallstones containing appreciable amounts of cholesterol could be successfully managed in this fashion. From one to six weeks might be required for the dissolution or fragmentation and passage of the stones. Various other men have used Pribram's technic or variations of it with varying degrees of success. No

one has been able to report such consistently good results. In 1953 Best[2] described his investigation of 113 agents with regard to their ability to dissolve gallstones. Ether and chloroform would appear to be the only effective substances yet employed. In Best's[1] opinion the best results were to be achieved by combining the solvent action of ether or chloroform with his biliary flush regime to help the spontaneous passage of the stone fragments.

Certain valid criticisms can be made of this method of dealing with overlooked common duct stones. It is time consuming for both patient and doctor; it is painful for the patient and, in the hands of most practitioners, has been found to be completely unreliable. At our own clinic in Buffalo this method has been attempted on several occasions, but with only an occasional satisfactory result. Those of you who have had experience with this therapeutic method I am sure will agree with the statement that it would be better if the occasion for its use did not come about.

3. Operative Cholangiography. Operative cholangiography was first described by Mirizzi[12] in 1932 and affords the most effective means at our disposal to prevent stones in the common duct from being overlooked at exploration. Despite the fact that Mirizzi and others have recommended the adoption of this procedure again and again during the past 20 years, surgeons by and large have been slow to accept it. Just why this should be is hard to understand. In this country, Mixter[13] of Boston has advocated its use most convincingly, but even now it is never employed in some clinics and more than occasionally in only a few.*

Two purposes may be served by operative cholangiography:

1. To determine the presence or absence of stones or sludge in a common duct prior to opening it in case the indications for its exploration are equivocal.

2. To exclude as certainly as is possible the presence of an overlooked stone after exploration of the common duct and the insertion of a T tube before the abdomen is closed.

The details of technic for operative cholangiography vary from hospital to hospital and are unimportant if certain principles are observed. The quantity of contrast medium used should be proportional to the size of the duct. The head of the patient should be lowered slightly below the horizontal to ensure that the upper reaches of the hepatic ducts are visualized. A cassette holder with Bucky grid should be placed on the operating table with the patient accurately positioned prior to the operation. Close coordination of the surgeon, anesthetist and roentgenologist at the moment the roentgenograms are taken is essential inasmuch as the anesthetist must momentarily stop the patient's respiratory movements when the contrast medium is injected and the roentgenographic exposure is made.

At the Buffalo General Hospital 20 to 40 cc. of 35 per cent Diodrast solution is slowly injected through the T tube after it has been securely fastened in place in the common duct. Two reontgenograms are taken: one, during the injection

* C. W. Clark of Syracuse, New York has recently surveyed by questionnaire 117 surgeons to determine their opinions as to the value of operative cholangiography. Seventy-eight surgeons used the procedure; 39 did not use the procedure. The usefulness of the procedure was evaluated as "excellent" by 68 surgeons; as "good" by 4 and as "fair or useless" by 39.

after approximately two-thirds of the quantity of solution to be used has been injected and a second, immediately after the injection is completed. On the basis of these films one can judge quite accurately whether or not the exploration has been complete and whether all calculi have been removed prior to closing the abdominal incision. In those instances in which the surgeon is not certain whether the duct should be explored or not, a similar amount of contrast media is injected into the common duct by means of a needle inserted through its wall or by means of a fine catheter or canula inserted through the cystic duct. Mixter[13] has pointed out the advantages of such a maneuver and there can be little doubt that when judiciously employed, many explorations with their attendant increased morbidity can be avoided. Also, it is true that some stones now overlooked in unopened ducts would be detected.

It is easy to magnify the difficulties inherent in any new technic. Such is human nature. The difficulties in large measure, however, can be overcome. It is much more difficult to overcome the inertia of some surgeons. Reported series would indicate that operative cholangiography increases the operating time by 10 to 15 minutes and that satisfactory films can be obtained in 85 to 90 per cent of patients.

Technical difficulties and associated pathology may from time to time contraindicate the use of this procedure. In Swedberg's series,[16] the largest reported to date, operative cholangiography was not feasible in approximately 6 per cent of patients for these reasons. Acute inflammatory processes involving the gallbladder, the bile ducts or the pancreas have been considered by all writers to be definite contraindications. Increased sensitivity of the patient to iodine also must be considered.

Despite expressed fears to the contrary, operative cholangiography does not seem to be associated with any appreciable risk. About 20 per cent of patients will have a portion of the pancreatic duct, as well as the extra hepatic ducts, outlined on the roentgenogram. No cases of pancreatitis or cholangitis are thought to have been caused by the injection of 35 per cent Diodrast solution in any report with which we are familiar or in our own clinic. Hay,[6] however, has found that 90 per cent of patients when subjected to cholangiography have a transient elevation of the blood amylase content if the pancreatic duct is outlined on the cholangiogram.

EXPERIENCE WITH OPERATIVE CHOLANGIOGRAPHY AT THE BUFFALO GENERAL HOSPITAL

Table III indicates the increase in the use of operative cholangiography at the Buffalo General Hospital during the past nine years. Some of the members of the Surgical Staff still fail to find any need for it but each year the number grows smaller. With increased use of the procedure has come an increase in the facility of taking the roentgenograms and of interpreting them. In some cases more than one set of roentgenograms are taken before both surgeon and roentgenologist are satisfied that the common duct has been adequately demonstrated.

Great reliance can be placed on the operative cholangiogram that is interpreted

TABLE III

Increase in the use of operative cholangiography at the Buffalo General Hospital

Year	Patients in Whom Common Duct was Explored	Patients in Whom Operative Cholangiography was Employed Incident to Common Duct Exploration
1945	35	2 (6%)
1946	27	2 (7%)
1947	25	2 (8%)
1948	30	6 (20%)
1949	30	10 (33%)
1950	33	11 (33%)
1951	28	7 (25%)
1952	63	22 (35%)
1953	66	38 (58%)
Total		100

by a competent diagnostic roentgenologist as being *clear*. In our series of 45 consecutive films so read a stone was found later in only one instance. This is an incidence of *false negative* interpretation of approximately 2 per cent. On the other hand, the incidence of *false positive* interpretations has been considerably higher. In 23 consecutive patients in whom *stones* were reported as being present on the operative cholangiogram, 4 patients were shown by further exploration and a postoperative cholangiogram not to have a stone. *False positive* interpretations therefore, were given in 17 per cent of cases. Occasionally the operative cholangiogram will demonstrate an obstruction at the lower end of the common duct the cause of which cannot be determined with certainty from a study of the films by themselves. In most instances such obstructions are due to a spasm of the sphincter of Oddi but in our series of 23 such patients, about one-third of the

TABLE IV

Interpretation of operative cholangiograms at the Buffalo General Hospital 1945–1953 (inclusive)

Number of Cases	X-ray Interpretation	Actual Situation
45	"Clear	44—clear 1—stone
23	"Stone"	18—stone 4—clear 1—not explored
23	"Obstruction"	8—stone 13—spasm of sphincter 1—blood clot 1—no postoperative check
9	"Unsatisfactory"	8—clear 1—no postoperative check
Total 100		

obstructions were due to remaining stones. In the total experience at the Buffalo General Hospital since 1945 the incidence of unsatisfactory films has been 9 per cent (table IV).

Table V indicates that there is a surprisingly large number of stones overlooked at our Hospital during exploration of common ducts that contain calculi; a much larger number than had been expected when this study was started. Similar studies in other clinics probably would be equally surprising. It is likely that the lower incidence of overlooked stones in 1952 and 1953 is due to the increasing use of operative cholangiography. It will be noted that, of 57 choledochostomies with exploration in which stones were overlooked, only 12 were done with the help of operative cholangiograms (table VI). In this group of 12 patients, therefore, it might be said that operative cholangiography as a method failed to achieve the end desired. Such is not the case, however. The roentgenogram taken during operation was completely misleading in only one instance, a patient in whom a large amount of contrast media, injected into a large dilated duct, completely obscured a single small remaining stone. In 4 other patients, the roent-

TABLE V

Overlooked common duct stones, Buffalo General Hospital
1945–1953 inclusive

Year	Choledochostomies with Stones in Duct	Choledochostomies with Stones Left in Duct	Percent of Stones Left in Duct
			%
1945	25	7	28
1946	13	4	31
1947	19	5	26
1948	25	8	32
1949	18	5	28
1950	26	9	35
1951	12	4	33
1952	36	8	22
1953	32	7	22
Total	206	57	28

TABLE VI

Analysis of use of operative cholangiography to overlooked common duct stones, Buffalo General Hospital
1945–1953 inclusive

Choledodochostomies with overlooked stones—57
1. Choledochostomies without operative cholangiography—45
2. Choledochostomies with operative cholangiography—12

Operative cholangiogram
"Clear"—1
"Obstruction"—4
"Stone"—7

genogram was interpreted as showing *obstruction*. In each instance it was assumed by the surgeon that the obstruction was due to a spasm of the ampulla of Vater. The possibility of such obstructions being due to stones still retained has been mentioned previously. In each of the remaining 7 patients the operative cholangiogram was reported to the surgeon as showing a stone or stones. In these patients the surgeon either chose to ignore the roentgenographic evidence or thought that the condition of the patient did not justify further exploration. In most instances, however, these cases represent a refusal on the part of the surgeon to believe the interpretation of the roentgenogram which did not confirm his clinical appraisal of the situation arrived at by the usual methods of examination and exploration. As the surgeon gains experience with operative cholangiography and appreciates its value as well as its limitations, he learns to take more seriously the positive film, for there is at least a 4 to 1 chance that the film is correct and that his clinical estimation is wrong.

SUMMARY

There is relatively little information available as to the incidence of overlooked stones in the common duct after the usual routine exploration, but it probably averages 20 to 30 per cent.

The overlooked stone may be removed surgically when it becomes evident or produces symptoms. Attempts may be made to produce its dissolution and fragmentation by the injection of chemicals. Its incidence possibly can be decreased by the use of operative cholangiography.

At the Buffalo General Hospital during the past year there has been a marked increase in the use of operative cholangiography. At the same institution during the past nine years, 206 explorations of the common duct containing stones have been performed. In 57 instances stones have been left in the duct after exploration. In only 12 of these patients was operative cholangiography done. The apparent failure of operative cholangiography in these 12 patients was due to limitation of the method itself in only 5 instances.

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A REVIEW OF PRESACRAL TUMORS AND REPORT OF A CASE OF PRESACRAL DERMOID CYST

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Presacral tumors are tumors which originate anterior to the sacrum and the coccyx and are of rare occurrence. Calbet[4] has reported that congenital tumors of the sacrococcygeal region occur only once in 34,582 births. One such tumor, a presacral dermoid cyst, was seen and treated by us on the Surgical Service at the Tampa Municipal Hospital about 18 months ago. This case will be presented in this paper.

The tumors in the presacral region are of several types and have been divided by Dockerty[6] into four classes, as follows: (1) congenital anomalies, (2) bone tumors, (3) neurogenic tumors and (4) a miscellaneous group. The tumors most commonly found in this region are dermoids, chordomas, ependymomas, teratomas, Ewings' tumors, giant cell tumors, neurofibromas and fibrosarcomas. The malignant tumors are chordomas, ependymomas, fibrosarcomas and metastatic lesions from primary growths elsewhere. Ewing[5] states that congenital presacral anomalies arise from three primary germinal layers and divides them into teratoids, tumors which are composed of various types of tissues but not arranged in a definite organ and true teratomas which are fully formed organs not related to the neighboring structures.

The embryonal structures which give rise to dermoid cysts in the sacrococcygeal region are (1) the fovea coccygea and the coccygeal vestiges of the neural canal, (2) the neurenteric canal, (3) the postanal gut, (4) the proctodeal membrane, or (5) from ectodermal inclusion of cutaneous surfaces.

Dermoid cysts are the most common of all types and may be found both anterior and posterior to the sacrum, close to the coccyx or deep in the hollow of the sacrum and in the ischiorectal and perirectal areas, but usually close to midline. (Dermoid cysts are ectodermal in origin with a lining of flat squamous epithelium, which together with mucous glands in the cyst wall result in continuous desquamation and secretion of mucoid material.) Spina befida may be found in association with this congenital group of tumors, but usually there is no direct connection between the two. Normally dermoid cysts do not manifest themselves until adult life—the majority of patients falling between the ages of 20 and 50. However, there are a few case reports in which the tumor was observed at birth. Dermoid cysts usually are encapsulated and may compress but do not invade adjacent structures. Thomason[17] has emphasized that openings in the sacrococcygeal region usually are the external manifestations of cysts or tumors of developmental origin. Whittaker and Pemberton[18] in a study of 22 cases of presacral tumors reported that, of these 22, 9 were dermoid cysts. These

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tumors appeared chiefly in women. Dermoid cysts in the presacral region have been reported as occurring only in females, usually in the childbearing period. Manheim, Druckerman and Peskin[14] have mentioned only one dermoid cyst found in the presacral region in 97,000 admissions to their hospital. Lahey[12] reports 3 cases from the records of the Lahey Clinic—all of females. Lahey and Eckerman[12] stated that presacral dermoid cysts should be suspected in patients who have sinuses and abscesses about the anus and who have had repeated operations without cure. Ewell and Jackson[7] have reported an infected presacral dermoid cyst which perforated the vagina producing an admission complaint of vaginal discharge.

Symptoms produced by early and small growths in this region usually are negligible, whereas, those caused by large tumors may be pronounced and disabling. Symptoms are not modified materially by the pathologic histology of neoplasms in this region because the chief symptoms of tumors in this region are usually the direct result of mechanical pressure which produces pain and anesthesia of varying degrees. Digital examination of the rectum is the most essential part of the physical examination and these tumors usually are palpable by rectal examination. The sacrococcygeal chordoma is a tumor of low grade malignancy and is derived from remnants of the primitive notochord. The name chordoma was first applied to this growth by Ribbert[16] in 1894. The chordoma is characterized by slow growth; by a tendency to invade bone; by local recurrence after surgical excision and by the fact that it exhibits only a slight sensitivity to irradiation. A chordoma seldom metastasizes to distant lymph nodes. The first complete description of a sacrococcygeal chordoma was made in 1900 by Hundling,[11] who discovered it while in the process of performing a postmortem examination on a 7 month old stillbirth. The occurrence of this tumor is rare. During a 13 year period in Memorial Hospital in New York, where 13,000 cases of cancer were seen, there were only 7 recorded cases of sacrococcygeal chordoma.[10] It has been stated that trauma may play a part in the development of these tumors. This tumor has been described as occurring at any age from 3 months to 78 years of age with the median age being 46 years. In contradistinction to dermoid cysts of this region, chordoma occurs more frequently in males than in females. Although the most frequent site is the sacral region, chordomas may occur at any level along the vertebral column. The second most frequent site is the sphenooccipital region. Gentil and Coley,[10] in 1948, reported 135 cases of sacrococcygeal chordoma following a complete review of the literature.

The most common early symptom experienced by a patient with sacrococcygeal chordoma is pain, which at first is mild but becomes more severe with extension of the tumor to involve the nerve roots and the sacrum. As the tumor spreads to involve the rectum or the bladder, constipation or urinary difficulties will occur. In the later stages fecal and urinary incontinence may develop and in this advanced stage the involvement of nerve roots produces motor and sensory changes of the lower extremities, the gluteal region and the genitalia. The tumor may extend anteriorly or posteriorly. If there is a posterior extension a

visible and palpable tumor usually will result. If anterior extension occurs, only rectal palpation will reveal the tumor. Mabrey[13] has found that patients with sacrococcygeal chordomas have had symptoms for three years before seeking medical aid. This fact emphasizes the fact that many of these tumors could be found on routine rectal examination long before progressive growth produced symptoms. In a case reported by Ewell and Jackson,[7] the presenting symptom was recurring pain in and about the rectum and vagina followed by discharge of pus by way of the vagina.

Chordomas are soft tumors. Histologic examination usually reveals rounded cells growing in cords and having vacuoles in their cytoplasms and/or nuclei and are accompanied by an abundance of mucinous material. They have been described as resembling the structural material found in the nucleus polposus of the spinal column. Chordomas are slow growing and have a low degree of cellular activity. As the tumor develops there seems to be a predilection for extension of the growth along the course of the blood vessels and to adjacent muscles and nerves, with invasion of the bones of this region rather than a spread of the disease by way of the lymphatic system. The rectum and perineum seldom are invaded by the neoplasm which very rarely metastasizes until late in the disease. The most discouraging factor in the treatment of chordomas is the marked tendency of recurrence, chiefly due to the difficulty of complete primary extirpation of the disease.

Sacrococcygeal teratomas relatively are uncommon and less than 100 have been reported in the literature. This type represents only a very small percentage of the presacral tumors. These tumors invariably are thought to be present at birth and about 90 per cent are recognized at the time of birth. This has been reported as one of the causes of dystocia. Seventy-five per cent of patients with this type of tumor are females. The exact origin and classification of this type of tumor is in some doubt. Sacrococcygeal teratomas may be rudimentary organ masses representing an ill-developed pygopagus twin, or they may represent tissue which arises primarily from cells already present in the sacrococcygeal region during normal embryonic development such as the postanal gut or the neurenteric canal. During development of the fetus there is a very close approximation of nervous, intestinal, bony and connective tissue in this region. Teratomas of this region always originate retrorectally and are attached to the coccyx or to the sacrum. From here they may extend downward and posteriorly into the buttocks or they may extend upward behind the rectum into the abdominal cavity. About 15 per cent of sacrococcygeal teratomas develop malignant changes. Characteristically the growth of a teratoma parallels the growth of the child. Therefore, rapid growth may and usually does occur with this tumor even though it is benign. The treatment for this lesion is complete excision.

The treatment of presacral tumors is excision with or without radiation therapy. The tumors in this region as a rule are benign or are of low grade malignancy and therefore, a good result can be expected if a benign neoplasm is completely removed by surgical excision. Low grade malignant tumors should

not recur, when removed, if the line of excision is extended well beyond tumor tissue and the field is left free of contamination by malignant cells. Complete extirpation usually is possible with a dermoid cyst but is almost impossible with a chordoma. Cheskey[5] has stated that the prospect for complete eradication of a chordoma is extremely poor. All patients operated upon, except those seen in the most recent years, have died of recurrence. Whittaker and Pemberton[18] found that the evidence of recurrence is high and if local recurrence does occur it probably is due to incomplete removal of the original growth. Fletcher and associates[9] in 1935 reported 1 case of a patient who was apparently well seven years following operation without evidence of recurrence or metastasis. Treatment is aimed at complete excision of the tumor which usually can be achieved by a posterior approach and removal of the coccyx with the patient in the Kraske position. Radiation therapy is indicated following the excision of the malignant variety of tumors. It generally is agreed that teratomas in the newborn should be excised in early infancy to avoid deformity, dangers of bowel obstruction and malignant changes.

The treatment of presacral ependymomas requires an extensive laminectomy as this tumor tends to invade the foramina and frequently will extend up the lumbar canal. However, despite this tendency to extension, Adson[1] has pointed out that, as a whole, ependymomas are extremely slow growing and frequently can be completely enucleated and an excellent prognosis given. In the treatment of malignant tumors, the dissection may be extended up to include as much of the sacrum as is necessary to the sacroiliac articulation. The majority of chordomas of this region occur in the lower part of the sacrum and the coccyx and therefore are usually accessible for removal. Mixter and Mixter[15] have stated that a wide excision of bones, muscles and nerves of this region should be done if necessary for complete eradication of the disease. They have shown that the segment of sacrum below the sacroiliac articulation together with the coccyx and all of its sacral nerves, except the first and second, can be removed without severe disability being incurred by the patient. Bowers[2] states that wide radical excision seems to be logical; although the structures are important, they are not essential to life and well being. Brindley[3] states that malignant lesions that recur are practically incurable surgically so every effort should be made to eradicate the tumor at the time of the initial operation. He further advocates the use of the cautery knife followed by thorough baking of the apparently diseased bone.

CASE REPORT

A 25 year old white woman was admitted to the hospital on April 21, 1952. Her chief complaint was increasing difficulty in moving her bowels. In fact, at the time of admission she was taking daily enemas in order to obtain bowel movements. She also stated that for approximately the past year she had noted a feeling of pressure in the pelvic region, which seemed to extend anteriorly in the region of the vagina.

Physical examination. The patient was a well developed, well nourished white woman. She was 5′4″ in height and weighed 145 pounds. General physical examination revealed no abnormalities except that found on rectal examination. On digital examination of the

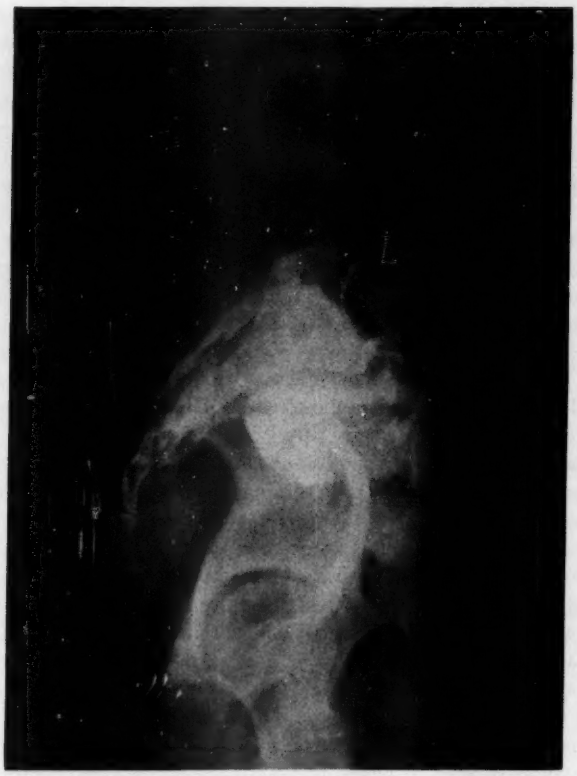

FIG. 1. A barium enema revealing anterior compression of the rectum by a presacral dermoid cyst.

rectum there was a nontender mass posterior to the rectum. The mass was soft and fluctuant to examination. There was no external evidence of gross abnormality in the region of the coccyx or anus. Proctoscopic examination revealed no evidence of abnormality of the mucosa of the rectum. A roentgenogram of the pelvis revealed sacralization of the transverse processes of the transitional type of the first sacral segment with a right sacroiliac synchondrosis producing scoliosis with convexity to the left. There was a congenital spina bifida of all the sacral segments. A barium enema revealed a large mass situated posterior to the rectum and compressing the rectum anteriorly. There was a slight irregularity of the rectal and proximal sigmoidal folds. Prior to surgery the bowel was prepared by administration of antibiotics orally for four days and this medication was continued postoperatively during her hospital stay.

Operation. Under general anesthesia a midline posterior incision was made extending from the sacrococcygeal junction distally to about 2 cm. from the edge of the anus. The coccyx was exposed and removed by separation of the sacrococcygeal ligaments. This exposed a large multiloculated cyst which was located posterior to the rectum and which seemed to almost entirely occlude the rectum. The cyst extended laterally on each side of the rectum so that it formed an encircling mass enveloping each side and the posterior portion of the rectum. The cyst seemed to be rather densely adherent to the rectum on its

posterior and lateral walls. The cyst was easily freed from the posterior and lateral walls of the pelvis. This cyst was dissected free with blung and sharp dissection and without undue difficulty. There was slight oozing at conclusion of the operation and a rubber tissue drain was left in the incision, following which, the wound was closed.

Pathologic Examination. The specimen consisted of a portion of lower end of the coccyx measuring 3½ by 2.2 by .6 cm., also a thick dense cyst wall lined by rough yellow membrane and measuring 3 cm. in diameter. Also present were several other similar cysts. The section showed a cyst wall partially lined on the inner surface by squamous epithelium. The epithelial cells showed desquamation into the lumen. The lumen contained a number of monocytes filled with lipoid material. In the wall of the cyst there were accumulations of monocytes and foreign body type giant cells. Focal infiltrations of lymphocytes were also present. The wall was made up, for the most part, of dense fibrous connective tissue. Pathologic diagnosis: benign dermoid cyst.

The patient's postoperative course was uneventful. The wound was dressed on the third postoperative day and the drain removed. There was only a smal quantity of serosanguineous discharge. The patient was discharged on the sixth postoperative day, and she was followed in the office. Skin sutures were removed on the ninth postoperative day. The incision was well healed. Rectal examination revealed a small degree of induration in the presacral region, but at this time she was free of her admission complaint of pelvic distress and constipation. She was last seen six months after operation, at which time she had completely recovered and rectal examination revealed no evidence of mass.

SUMMARY

A brief review of tumors occurring in the presacral region has been presented, including incidence, characteristics of the more common presacral tumors, and present treatment.

A case of dermoid cyst which was present in a 25-year old woman, producing a mechanical disturbance of bowel function, has been presented, with discussion of its diagnosis and treatment.

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SYMPATHECTOMY FOR VASCULAR DISEASE OF THE EXTREMITIES, WITH EMPHASIS UPON THE EXTENT OF THE OPERATION

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The subject of sympathetic denervation of the arms and legs by surgical measures has become of increasing importance during the past few years as there is little doubt that sympathectomy is helpful in many disease entities affecting the extremities. The results following surgical sympathectomy of the lower extremities have been much more satisfactory than those accompanying operation on the upper extremities. For this reason the principal part of the present discussion will concern the upper extremities. Many vascular diseases in particular can be benefited, provided an adequate sympathectomy can be obtained, but reports in the literature have noted recurrences of vascular tone in a distressingly large number of patients. These reports have indicated that recurrence to a significant clinical degree occurs in from 30 to 75 per cent of the patients.[1] A large number of theories have been proposed as to the possible cause of this return of vascular tone but some of these theories appear no longer to be tenable. These theories will be reviewed briefly.

Increased sensitivity of the arterioles of the hand to circulating adrenalin following a postganglionic sympathectomy has been cited as a cause for most recurrences. There can be little doubt that this theory has retarded the progress of developing adequate sympathectomy for the upper extremities more than any other single factor. There now appears to be sufficient information indicating that, although some increased sensitivity to circulating adrenalin does occur, this sensitivity will follow any type of sympathectomy and is not of major importance.[4] The recurrences usually appear when such hypersensitivity would be diminishing, or would have disappeared.

A second theory regarding the relapse following sympathectomy concerns the possibility of a local fault in the arterioles of the extremities. This theory has been expounded by Simmons and Sheehan[5] and apparently does take place in a very small minority of the patients. In these instances the local fault of the blood vessels is so severe that it cannot be overcome by any vasodilatation following sympathectomy. In the present series of cases there is one failure that may be attributed to this local fault, although it is probable that the sympathectomy itself was not of sufficient extent. Simmons has shown conclusively, however, that such a local fault in vessels can occur as the vascular changes persist in spite of sympathectomy and blocking of the peripheral nerves.

Another theory regarding the cause of recurrences is that of regeneration of

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sympathetic fibres. There certainly is a great deal of evidence indicating that regeneration can, and does occur, in the sympathetic system to a much greater degree than in the somatic nervous system. There is one report in the English literature in which the author states that a splanchnic nerve had so completely regenerated that it resembled the nerve on the opposite side and he believed that the nerve also was functionally intact. There are no other instances available in which regeneration can be definitely proved, although it does appear that it can occur in a large number of cases. It is difficult, however, to believe that regeneration is the sole cause for such recurrences as it does not occur to a significant degree in the lower extremity. It would seem just as probable that the sympathetic nerves would regenerate going to the lower extremity as to the upper extremity and, therefore, it does not appear that regeneration of the fibres themselves can be the sole cause for the clinical relapses.

Another possible cause for the recurrence of vascular tone following sympathectomy, is the probability that the original sympathectomy itself was not adequate in extent.[2] Knowledge of the anatomy of the sympathetic fibres going to all of the extremities is increasing daily and certainly many of the previous concepts shall have to be discarded. Some reports in the present literature still state that the sympathetic fibres to the upper extremity arise in the second and third thoracic roots and discard the possibility of fibres arising in other areas. There is an abundance of evidence indicating that the fibres for the upper extremity primarily arise in the second and third roots, but that some fibres may go to the upper extremity from levels as low as thoracic eighth and as high as the midcervical region. In addition to the fibres that pass from the anterior roots into the sympathetic ganglia, there now is abundant evidence that some sympathetic fibres do not go through the ganglionated trunk and, in fact, have small ganglia incorporated in the nerves themselves. These ganglia have been well demonstrated by Skoog.[6] Such sympathetic fibres would not be interrupted by complete removal of the entire ganglionated chain and could be removed only by section of all of the appropriate nerves. The existence of such fibres also going to the lower extremities has been shown by Ray and Console,[3] and they have demonstrated that a complete denervation of the lower extremity will not be obtained unless the ganglionated chain is removed extensively and the twelfth thoracic, first and second lumbar anterior roots, also are sectioned. The fibres that traverse these three roots, however, are not of major clinical importance in the vast majority of cases.

The operation that is here advocated is one that will denervate the upper extremity in as great a degree as possible. The posterior approach is used and sections of the third and fourth ribs are removed. The second and third, and occasionally the fourth, intercostal nerves are removed. This is done by tracing the nerves back to their exit from the dura and actually doing intradural section of the anterior and posterior roots. The basis for this part of the operation is the known fact that many fibres and, in fact, the majority of fibres, going to the upper extremity traverse these two nerves, and there is a strong possibility that some of the sympathetic fibres do not traverse the ganglionated chain.

These fibres can be obliterated only by intradural section of the anterior roots. Such a section also mitigates against regeneration of the sympathetic fibres. The ganglionated chain then is reached as far down as possible, which is usually at approximately the seventh thoracic segment. The entire chain is removed up through the stellate ganglion. Visualization is excellent when the two ribs are removed. The entire ganglia themselves should be removed rather than any attempt at a preganglionic section. The operation easily can be done on both sides at the same sitting and the patient is therefore spared two operative procedures. There remains the possibility that some fibres are traversing the first thoracic nerve, but it is not believed that this nerve should be sectioned. In many instances section of the first thoracic nerve will cause a partial ulnar nerve paralysis and result in a crippling deformity of the hand. If vasomotor tone still remains in the hand following this operation it probably would be well to remove the middle cervical ganglion at a later stage, but this will be necessary in only a small number of cases. A Horner's sign will result from this operative procedure, but it is of no major consequence when one considers the severity of the disease for which the operation is done and also the fact that a bilateral Horner's syndrome is not markedly disfiguring.

With regard to denervation of the lower extremity, a muscle-splitting incision is used. With adequate relaxation it is entirely possible to remove the first, second, third and fourth lumbar ganglia through this incision. In all instances in the present series this operation has produced adequate sympathectomy of the lower leg. In some patients it may be necessary to resect the twelfth thoracic ganglia and also the anterior roots of the twelfth thoracic, first and second lumbar nerves, but this operation has not been needed in this group of cases.

The results in the type of lumbar sympathectomy that is here advocated have been published repeatedly and need no further emphasis. It is sufficient to state that the sympathectomy itself has seemed adequate in 38 patients, whose cases have been followed. Four of these patients ultimately have needed amputation, but they were all instances of far advanced vascular disease and the sympathectomy itself seemed to be of sufficient extent. It is worthy of mention, however, that sympathectomy may accelerate the process of gangrene if it already has begun in the toes. If the patients do well for the first few weeks following operation—if they have had gangrene—they appear to show improvement, but there is a definite possibility that early gangrene will be accentuated. This process well may take place because of a shunting of blood from the arterial to the venous system above small thrombosed arterioles.

The final results of the type of sympathectomy advocated here for sympathetic denervation of the upper extremity cannot as yet be stated. The possibility of recurrences cannot be ruled out for a period of at least two, and probably five, years. This type of operation has now been done for over four years and there seems to be a significant decrease in the instances of return of vascular tone. In a series of 14 surgical sympathectomies of the upper extremity there has been only one marked clinical failure and this occurred early after operation. In this patient the stellate ganglion was left in place. It should have

been removed. It is possible, however, that this case is one of local severe fault in the arterioles and even removal of the ganglion would not have produced a good effect. In 2 other patients there has been some slight return of vasomotor tone, but it has not produced any clinical symptoms. In two instances the classical operation of preganglionic sympathectomy was done on one side, and on the other extremity the extensive operation was done with removal of the ganglia from the stellate down through six ganglia. The second and third intercostal nerves were also removed. In each patient there has been a definite return of clinical vasomotor change on the side of the preganglionic sympathectomy, whereas, on the side of the extensive ganglionectomy there has been no return of sympathetic function.

The anterior approach has been used in very few patients because of the fact that the second and third thoracic nerves cannot be resected and it is believed that this is an integral part of the operation. There have been no instances in this series of long standing pain following the posterior approach, and this procedure enables one to obtain a much wider exposure and permits removal of more of the factors that probably contribute to the large number of recurrences.

SUMMARY AND CONCLUSIONS

The principal purpose of the present paper is to strongly advocate extensive removal of the sympathetic structures innervating the extremities when such operation seems indicated. In all of the cases studied in this series the patients have been given the various vasodilator and ganglion-blocking agents without permanent improvement. In a few instances it would appear that some of the patients have been made definitely worse by the use of these agents and it is apparent that the *borrowing-lending principle* of hemodynamics will may have a part in these bad effects. In some instances of vascular disease of the upper extremities, it would appear that there is a pronounced local fault in the vessels which cannot be overcome by sympathectomy but this occurs rather rarely. The possibility of hypersensitivity of the arterioles resulting from a postganglionic sympathectomy has been studied previously by many workers and does not contribute to the recurrences of vascular tone. The possibility of regeneration of sympathetic fibres being responsible for such recurrences is present, but it is not logical to presume that this is the only cause for such recurrences. In addition, it is difficult to understand why there is not more regeneration of fibres going to the lower extremity. It would appear that the reason for the better results following sympathectomy of the legs is that the classical operation actually does a more adequate sympathetic denervation of the lower extremities. In many instances it is probably impossible to completely denervate the sympathetic supply to the upper extremity without causing neurological damage, but in the majority of patients a widespread sympathectomy from the stellate ganglion down through the sixth thoracic segment, coupled with resection of the second and third, and possibly the fourth thoracic anterior and posterior roots, will produce excellent results. A further report will be made at a later date when the patients can be assessed after a minimum period of two years

following operation, but for the present it is believed that the extensive operation affords the best opportunity for good clinical results in patients requiring sympathectomy.

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SPONTANEOUS PNEUMOTHORAX

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Spontaneous pneumothorax is an emergency problem. It has the same mortality rate as ruptured appendicitis or lobar pneumonia. Treatment, once the diagnosis has been confirmed by roentgenogram, has been symptomatic. The patient usually is placed at bed rest. Oxygen is given if respirations are rapid or if cyanosis is present. If tension is present needle aspiration is performed. In the last few years I have been impressed by the difference in recovery rate; complications seen; and length of hospital stay in patients treated more actively.[2, 3] Active treatment consists of a closed thoracotomy made with a large needle or trocar through which a small catheter is placed in the pleural space and tightly sealed under water. I have collected the last 75 consecutive cases of spontaneous pneumothorax for comparison of the above effects of treatment.

Is there any other situation where an organ is allowed to lie useless and effect its own recovery when methods are at hand to influence almost immediate recovery to full function within the space of a few hours? Any surgeon doing an open thoracotomy for segmental removal or lobectomy tries to effect complete re-expansion of the remaining lung at the time of operation or soon after, by catheter suction. Is any pathology which spontaneously allows air to leak into the pleura ever as extensive as the raw surface left after a segmental resection? As this surface normally will seal itself over into an air-tight state in a very short time, I have reasoned that the pathologic conditions which cause pneumothorax spontaneously can be more properly treated with the lung expanded and functioning, and with fewer complications, than if allowed to run its own course.

This series consists of 75 consecutive cases of which there were 62 males and 13 females. The ages range from 12 to 82 years, the most common occurrence of pneumothorax being at 19 years, at 24 to 30 years, and 56 to 58 years. It was not clear in the history of a few, but 8 occurred during acute illnesses such as upper respiratory infection, bronchiectasis, asthma, bronchitis or tuberculosis; 28 occurred during mild, or moderate to severe exercise; 33 occurred while quiet, either in bed or sitting still. Of the 75, 9 had second attacks. The extent of collapse had no bearing on the disease state.

There were five deaths or a mortality rate of 6.7 per cent.

Case 1. A woman aged 30 was admitted to the hospital with 70 per cent pneumothorax which elaborated a great deal of fluid with tension. She was treated by needle aspiration, but died on the fifth hospital day.

Case 2. A woman aged 43 had 100 per cent tension which occurred immediately postoperative for minor gynecologic procedure which was performed under spinal anesthesia. Death occurred at time of diagnosis.

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Case 3. A man aged 62 was admitted to the hospital. Death occurred during a febrile pulmonary illness with pneumothorax producing tension and coma. He was relieved by needle aspiration but he died within 24 hours.

Case 4. A man aged 69 was admitted to the hospital with 50 per cent pneumothorax. His treatment was rest and oxygen. He died the third day in congestive heart failure.

Case 5. A man aged 82 was admitted to the hospital. His treatment was bed rest and oxygen. He died in 24 hours.

The length of hospital stay was from 1 to 91 days. In those patients in whom an interpleural catheter was used and placed under water seal—and there were 19 in the series—the average length of hospitalization was seven days. Complete expansion was obtained most often at the end of 48 hours or at the end of five days. The shortest length of time to complete re-expansion was 24 hours, the longest was nine days. In those patients treated without catheter, complete re-expansion was obtained in 1 patient in 72 hours. The majority could be followed only to the time of their discharge from the hospital but were expanding at that time, which was most often on the twelfth, nineteenth and twenty-second hospital day. The average was 15 days to discharge condition, i.e., re-expanded sufficiently to be ambulatory in most cases. Complete re-expansion was followed in certain patients requiring from 19 to 47 days, 1 of 71 days, 1 of 3 months, 2 of 4 months and 1 of 5 months. Should these unusual cases have been included the length of hospital stay would have been lengthened and hence would not be a true picture of the majority.

Only one complication occurred with catheter drainage—pleural fluid. Without the catheter, with all other methods of treatment, including needle aspiration, there were the following complications: pleural effusion, pulmonary edema, mediastinal emphysema, empyema, hemothorax—after repeated needle aspirations—cardiac decompensation, trapped lung, atelectasis and death.

The pathology showed there were 4 confirmed cases of tuberculosis and 4 suspected but nonconfirmed cases. Fifteen cases were proved by roentgenogram to have apical blebs after re-expansion. Six cases occurred during acute pulmonary inflammation. Five patients had obvious adhesions at the time of the first roentgenogram, 2 of whom showed intrapulmonary cavities adjoining the adhesions. Two patients had cysts within the lungs. Three patients had bullous emphysema with asthma. Thirty-one patients—all of the remainder—had no diagnoses as to the cause of spontaneous pneumothorax.

CONCLUSIONS

By the use of interpleural catheter with water sealed draining, the need for hospitalization may be cut in half.

Earlier diagnosis of the pathology present may be achieved.

All of the more serious complications possibly may be prevented with much shorter disability.

All deaths probably may be prevented by use of catheter at the time of diagnosis of spontaneous pneumothorax.

NOTE: The summaries, facts, and conclusions of this paper are based on the clinical material obtained from 75 cases and histories seen in three general hospitals.

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THE TREATMENT OF PERFORATED APPENDICITIS*

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No final word has been written on the surgical management of peritonitis due to perforated appendicitis, but the reduction of death rate as recorded by Chandler, Long and Ott[1] is good evidence that the present methods of treatment are exceedingly successful. Each case is an individual problem, but the following measures generally are applicable and generally are acceptable: (1) removal of the offending organ; (2) full dosages of antibiotics; (3) intravenous fluids and electrolytes; and (4) gastric suction when indicated. In addition, analgesics, blood transfusions, oxygen, and vitamins frequently are of importance. All of these are acceptable to everyone, but then comes the old argument between surgeons as to whether to drain or not to drain the peritoneal cavity. One of us (J. R. M.), from 1940 to 1945, used the nondrainage and castor oil treatment, so ably described before this association by Lee[3] in 1946. It was then discontinued because of the number of residual abscesses.

The purpose of this paper is to report cases of patients treated by the old method of drainage and to show statistically the number of major postoperative complications. We belong to the school who think that a rubber or penrose drain does no harm and that fewer postoperative complications are encountered in patients treated in this manner. It also is our opinion that the reason this treatment has become unpopular is because these drains were, first, wrongly placed and, second, that they were removed too early, apparently for fear of complications due to the drain.

Schullinger,[5] in 1947, in stressing the importance of drainage, made this statement, "Unfortunately, with the advent of antibacterial agents and better methods of treatment, it is tempting and misleading to overlook sound, fundamental principles of surgical therapy. These principles (of drainage) should be observed by surgeons of average ability, such as Dr. Jones, Dr. Smith, Dr. Brown and myself."

Whenever doubt exists as to a possible contamination, we do not hesitate to use drains, probably often unnecessarily but never with any regrets. A drain sometimes is used by us in gangrenous appendices, whenever necrotic tissue is present, following severe operative trauma, whenever the appendiceal stump is not securely ligated, and always when there is an actual peritonitis.

METHOD OF TREATMENT

Treatment consisted of prompt operation as soon as the diagnosis could be established and the patient could be properly prepared. All operations were

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FIG. 1

done through a McBurney type of incision and appendectomy was done on all patients except in three localized abscesses where drainage only was done. Penrose or cigarette drains were inserted, the number depending on the severity of the peritonitis. In an early rupture with early peritonitis only one drain was used, that being placed at the site of the removed appendix. Usually, however, especially in more extensive peritonitis or in pelvic or retrocecal appendices, two drains were used (fig. 1). One of these was placed in the pelvis and the other in the lateral gutter or the retrocecal area.

No antibiotic or sulfonamides were placed in the peritoneal cavity and the wounds were closed in layers about the drains. Antibiotic therapy has consisted mainly of the combination of streptomycin and penicillin. A few patients have been treated by terramycin, using it intravenously until it could be tolerated by mouth, and these patients have all done well. Both methods are satisfactory and we have no choice between the two.

The antibiotics should have a wide bacterial spectrum, as reported by Poth,[4] because of the intestinal flora, and should cause as few side reactions as possible, such as enteritis, nausea and other toxic symptoms. The combination of penicillin and streptomycin comes nearest to fulfilling these requirements and, in addition, can be given parenterally even in the presence of nausea and vomiting.

TABLE I

Perforated appendicitis

	Total	Abscess	Early Spreading Peritonitis	Fulminating Peritonitis
No. of cases	87	14	67	6
No. of complications	5	0	4	1
Mortality rate	1.15%	0	0	16⅔%
Avg. no. hosp. days	14.18	14.39	13.63	18.60

A disadvantage is that resistant strains will develop quicker with this combination than when using aureomycin or terramycin. Terramycin and aureomycin are best given by mouth but they cannot be tolerated when there is nausea and vomiting. They sometimes cause gastrointestinal disturbances. Until recently terramycin had to be given intravenously, thus requiring the presence of a house staff member for each dose and was a definite disadvantage. It now, can of course, be given intramuscularly and will eliminate this disadvantage.

Levine (gastric) tubes were not used routinely, being inserted only in frank gastric dilation and this rarely was necessary. The patients were kept on a starvation diet, consisting only of small quantities of water until nausea had ceased, which averaged about two days. They then were started on food which was increased from day to day.

The gaseous distension or ileus, which was mild except in 2 patients, was treated only with sedation and rectal tubes—rarely resorting to the use of prostigmine-like drugs—and usually a bowel movement occurred on the fifth postoperative day.

General supportive therapy consisted of blood transfusions, intravenous fluids, and always with a keen lookout for a proper electrolyte balance. In approximately one half of the patients early ambulation was employed.

The drains were not disturbed until the patient was afebrile, or nearly so, and then they were removed usually in stages. This removal was started on about the sixth or seventh postoperative day; but if the patient still had fever of any significance, drain removal was delayed indefinitely.

STATISTICS

The following are all cases of perforated appendicitis for the five year period from 1949 through 1953 in an 85 bed hospital.* All operations were done by one of four surgeons. Intraperitoneal drains were used routinely. There were a total of 87 such cases (Table I) and they are further subdivided into 67 patients with early spreading peritonitis, 14 patients with abscess formation, and 6 patients with generalized, fully developed peritonitis. One of the generalized peritonitis patients died, making a mortality rate of 1.15 per cent of all cases of ruptured appendices. This one death occurred in a patient who had malrotation of the cecum and had been treated at home for gallbladder colic for three days.

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TABLE II

Perforated appendicitis (complications encountered)

Pelvic abscess	1
Other abscesses	0
Fecal fistula	0
Paralytic ileus	2
Phlebitis	1
Overwhelming toxemia	1
Incisional hernia	0
Evisceration	0
Wound abscess	0

She was referred to the hospital with a diagnosis of a ruptured gallbladder, arriving in critical condition. Her death, although no autopsy was obtained, probably was due to overwhelming toxemia, although there were signs of a cerebrovascular accident manifested by pupil inequality and one sided extremity weakness.

The complications encountered (Table II) consisted of one pelvic abscess, two cases of moderately severe paralytic ileus, one phlebitis and one overwhelming toxemia. The one death most probably was due to toxemia rather than a cerebral accident. No fecal fistulas, incisional hernias, wound abscesses, evisceration or intestinal obstruction occurred as complications.

Both the early spreading and fulminating types of peritonitis were treated by the method previously described. Abscess formation received conservative treatment as long as the infection continued to subside, followed by elective appendectomy at a later date. Those which didn't subside had an operative drainage procedure, removing the appendix at the same time if it could be easily found, but if not easily found elective appendectomy later was done.

COMMENTS

In commenting on these cases it seems that in spite of the fact that we have improved postoperative care and antibiotics, that the indications for applying sound principles of surgery, such as drainage of the peritoneal cavity, are about the same as when first advanced by Yates[6] in 1905. This has been beautifully illustrated in 1953 by Fowler and Bollinger[2] who had 38.6 per cent complications in nondrainage cases as compared to 13.9 per cent in those drained. They also reported that the only cases in their series of evisceration and incisional hernia occurred in those not drained. Our statistics also illustrate the low rate of postoperative complications, having only 5 in 87 cases or 5 per cent, and 1 of these, namely the phlebitis, could occur as a postoperative complication for any disease, actually making it only four complications. We readily admit that our period of hospitalization is longer than in uncomplicated, nondrainage cases, but we prefer it this way rather than having more complications which could have been prevented by proper drainage.

Those opposed to intraperitoneal drains for peritonitis say that it is impos-

sible to drain the entire peritoneal cavity, which is certainly true. These drains affect only a small area, but that is the area of maximum danger and if properly placed they will provide means for purulent material to escape rather than pooling in this location.

SUMMARY

Eighty-seven consecutive cases of perforated appendicitis occurring over a recent five year period are reported; all of these patients were treated with intraperitoneal drainage. The number of complications were minimal, especially as concerns residual abscess, fecal fistula, wound infection, incisional hernia, evisceration, and intestinal obstruction. We believe strongly that these complications are much fewer than in any similar group treated without such drainage. Emphasis is placed on properly locating the drains and on not removing them too early, having observed no harmful effects from their presence even if left for long periods of time.

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ACUTE PNEUMOCHOLECYSTITIS

CASE REPORT

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Acute pneumocholecystitis is a descriptive term applied to an acute infection in the gallbladder in which gas also is present in the gallbladder. Pericholecystic infiltration with gas may or may not be present. A review of the literature gives the impression that this is a relatively rare condition. We have found 31 cases. It rarely is mentioned in textbooks on diseases of the gallbladder and biliary tract.

It is the purpose of this paper to report an additional case, which is typical of those previously reported. This case also is interesting because the gallbladder subsequently sloughed out entirely through the cholecystostomy drainage wound and cholecystectomy was not required.

Historically, in 1901, Stolz reported 3 cases discovered at autopsy, in which gas was present in the gallbladder and bile ducts, thinking this was a postmortem feature. It is doubtful if these were true cases of pneumocholecystitis. In 1908, Lobinger[4] reported a case in which gas was found in the gallbladder of a patient during a cholecystectomy. Since that time, additional cases have been reported periodically. Hegner, in 1931,[3] reported the first case in which the preoperative diagnosis was made by roentgenographic examination.

Acute pneumocholecystitis can be differentiated from acute cholecystitis only by roentgenographic evidence of gas in the gallbladder. However, the presence of gas alone in the gallbladder necessarily does not make the diagnosis.

As pointed out by Abel and Rousselot,[1] gas may gain access to the biliary tree in one of three ways: (1) Fistulous communication between the gallbladder or biliary tree and some portion of the gastrointestinal tract. The fistula results from an inflammatory process or is secondary to a surgical procedure such as choledochoenterostomy or cholecystoenterostomy. (2) Incompetence of the sphincter of Oddi, which may be intrinsic or secondary to a surgical procedure such as sphincterotomy. (3) Gas within the gallbladder due to infection by gas-forming organisms, i.e. pneumocholecystitis.

Gas in the gallbladder from the first two conditions usually can be differentiated from the third condition by roentgenograms, although occasionally a fistula cannot be demonstrated by roentgenograms. McCorkle and Fong[5] state that usually, when the gas in the gallbladder arises by communication with the intestinal tract, the cystic duct, hepatic ducts, and common duct are seen. The gallbladder itself is contracted, or is not seen at all. When the gallbladder can be seen, the gas is confined to the lumen. In an upright film, no fluid level can

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be demonstrated in the gallbladder. These authors stated further that the roentgenographic appearance of the circumferential ring of gas present in acute pneumocholecystitis may be simulated by lipomatosis of the gallbladder wall. The reason for the similarity is that lipomatous tissue, possessing a density less than that of surrounding viscera, may appear black against a light background on a roentgenogram just as gas does. In lipomatosis, however, the shadow does not change configuration with change in position of the patient as the gas shadows do; there is no gas-fluid level, and there is no hour to hour or day to day change in the roentgen picture.

Acute pneumocholecystitis gives a characteristic appearance in the roentgenogram of the abdomen taken in the supine position. Gas is present in and about the gallbladder wall and in the lumen. In an upright or lateral decubitus roentgenogram, gas often is demonstrated over a fluid level. Serial films show the gallbladder to be distended in the early stages; later, one sees that there has been absorption of the gas from the lumen; the gas becomes more streaked and irregular, and slowly decreases. The size of the gallbladder tends to decrease. The finding of bubbles of gas at an irregular distance from the wall of the gallbladder, or from the air-filled lumen of the gallbladder, indicates that the pericholecystic tissues are filled with gas. We believe that this characteristic picture of streaked *bubbly* gas within the wall of the gallbladder and the pericholecystic tissues is pathognomonic of pneumocholecystitis.

Bell and associates,[2] have given an excellent review of the previously reported cases and add 2 of their own making a total of 27. They found 21 cases in males, which is interesting because of the higher incidence of gallbladder disease in females. The age of the patients varied from 32 to 74 years, but the majority were between 50 and 70 years of age. Eight of these 27 patients had diabetes, so diabetics evidently have a predilection for pneumocholecystitis. Of an additional 4 cases reported in recent literature, 2 of the patients had diabetes as does our patient, making a total of 11 diabetics in 32 cases—an incidence of about 34 per cent.

The causative organisms, when isolated, have been Clostridium welchii, Bacillus coli, Streptococci, both aerobic and anaerobic, and Staphylococcus albus. No causative organism was isolated in several of the cases.

Treatment has varied from conservative medical to radical surgical. Chemotherapy, polyvalent vaccine, and roentgen therapy, alone or in combination with cholecystostomy or cholecystectomy, have all been used. Four deaths occurred in these 32 patients; three in the preantibiotic days. The fourth died from cerebral apoplexy. All of the deaths occurred in operative patients.

CASE REPORT

This patient, a 56 year old white man, a mine foreman, was admitted to the hospital with a chief complaint of abdominal pain. The pain had occurred rather suddenly in the upper abdomen with some radiation into the right lower quadrant. It had been a generalized type of pain with no localization at the onset. There had been severe nausea with repeated vomiting. He also had had a rather severe generalized type of headache with some slight vertigo. There was no past history of any indigestion. He apparently had been able to eat all types

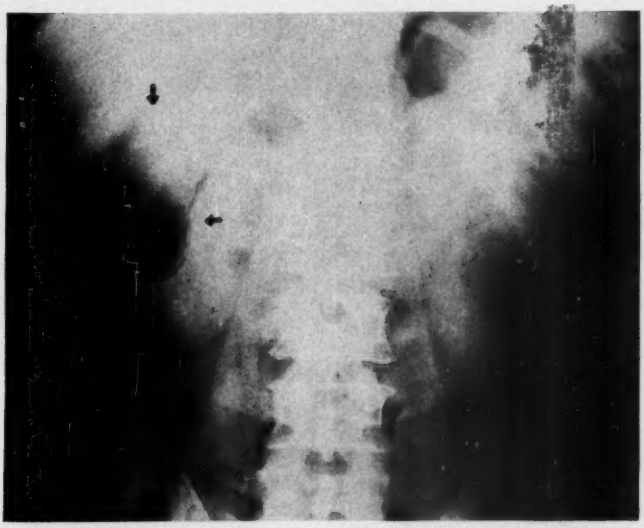

FIG. 1. Accumulation of gas outlining the gallbladder and also within the gallbladder

of food with no untoward results. His bowel habits had been regular. About one year prior to the present illness he had been found to have mild diabetes. Since that time he had done fairly well until about three months prior to his present illness at which time he developed some polydipsia and polyuria. He had never taken insulin.

On admission to the hospital the patient was having severe pain and was sitting in a doubled-up position. The blood pressure was 110/72, pulse 98, and temperature 101 F. He was well developed and nourished. The heart and lungs were normal. There was marked tenderness in the right upper quadrant of the abdomen with rigidity in the gallbladder area. The lower abdomen was soft, but there was some slight tenderness in the right lower quadrant. The gallbladder could not be felt. The white blood count was 19,200 leukocytes per cu.mm. with 81 per cent polymorphonuclear cells and 19 per cent lymphocytes. The urinalysis showed a 4 plus sugar with no acetone or diacetic acid present. The blood sugar was 208 mg. per 100 cc. A roentgenogram of the abdomen taken in the supine position (fig. 1) was reported as follows: "The right upper quadrant shows a gaseous accumulation apparently outlining the gallbladder wall and also lying within the gallbladder, which appears to be typical of acute pneumocholecystitis. The upright study (fig. 2) shows an air fluid level in the gallbladder with the gallbladder wall outlined by a thin layer of gas." The following day a tender mass 5 cm. in diameter could be palpated in the gallbladder area.

During the first week of hospitalization treatment was conservative. The diabetes was easily controlled by routine measures and small doses of insulin. He was given aureomycin in addition to frequent intravenous fluids. During this week the temperature varied from 100 F. to 102 F. He continued to have rather persistent pain in the right upper abdomen. Because of poor response to conservative treatment and the persistence of the mass, operation was done on the eighth hospital day. The day before operation a roentgenogram of the abdomen with the patient in the supine position was reported as follows: "The appearance of the gallbladder is about the same except that there may be some increase in the fluid content. In a lateral decubitus position a roentgenogram still shows air fluid level and gas within the wall of the gallbladder with none being demonstrated in the common duct" (fig. 3). At operation the gallbladder was found completely covered with omentum. When it was partly separated from the liver, free pus was encountered. The fundus of the gall-

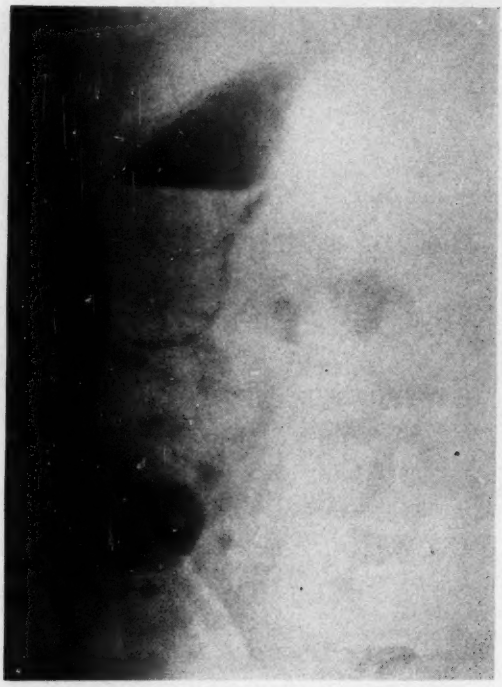

FIG. 2. Air with fluid level in the gallbladder

FIG. 3. Roentgenogram taken with the patient in the lateral position shows an air-fluid level and gas within the wall of the gallbladder.

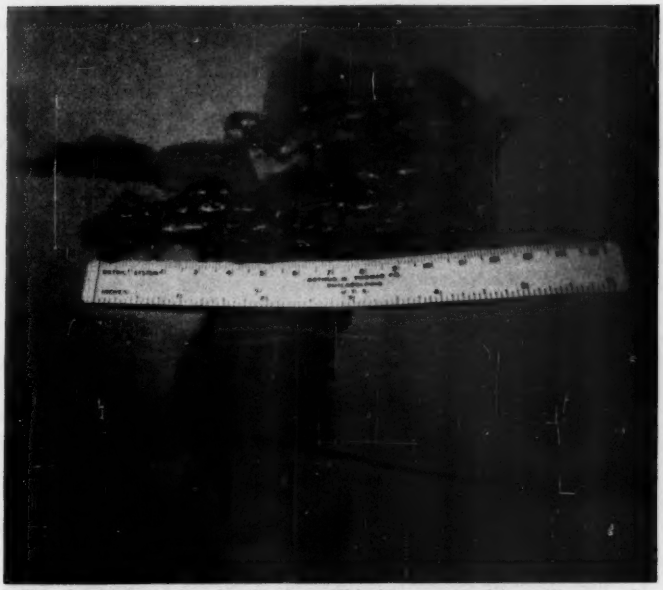

FIG. 4. Slough which appeared to be the gallbladder removed from the wound one month after operation.

bladder was visualized. It was gangrenous throughout and there were several small perforations which had been sealed by the omentum. No attempt was made to remove the gallbladder. It was aspirated and 30 cc. of air and approximately 40 cc. of dark brown fluid were evacuated. The gallbladder then was opened and a large quantity of black bilirubin particles were removed with a scoop. A section of the gallbladder was removed for biopsy. A large rubber tube was inserted into the gallbladder for drainage and tied in place with a purse-string suture of chromic catgut. A large rubber tissue drain was inserted down to the foramen of Winslow and brought out through the wound with the cholecystostomy tube. The day following operation the temperature returned to normal and remained so for the remainder of his hospital stay. The rubber tissue drain was removed on the third postoperative day. The cholecystostomy tube drained clear bile from the second postoperative day. The pathologic report of the biopsy of the gallbladder revealed nothing but necrotic tissue, heavily infiltrated with polymorphonuclear cells. Culture of the fluid revealed no causative organism, probably because of intensive antibiotic therapy. He was discharged on the fourteenth hospital day, seven days postoperative. The postoperative course was relatively uneventful. The drainage tube was removed from the gallbladder 11 days following operation. As an outpatient he was seen at weekly intervals. His course was satisfactory and there was minimal bile drainage from the wound.

Approximately one month following the operation, the patient was seen as an outpatient, with still some drainage from the wound. At that time a large slough was removed through the wound which appeared to be the gallbladder (fig. 4). Pathologic examination of this material was inconclusive due to complete autolysis. However, the configuration of the material strongly suggested a gallbladder. One week later, roentgenographic study was made and a gallbladder was not visualized. The patient again was admitted to the hospital approximately three months after the cholecystostomy. He stated that three days prior to this admission he had developed moderately severe upper abdominal pain accompanied by

nausea and vomiting. He noted a yellowish tinge to his skin and that his urine was dark in color and stools were clay colored. Examination on this admission revealed marked icterus. There was moderate tenderness in the right upper quadrant of the abdomen. The old operative wound was well healed. Laboratory studies indicated an obstructive type of jaundice. An operation was done two days after admission. At operation, a definite gallbladder could not be identified. A remnant of cystic duct was present. The common duct was enlarged, edematous, and covered with inflammatory adhesions, consistent with an acute choledochitis and cholangitis. The common duct was explored and calculi were not found. The duct was patent throughout as was also the sphincter of Oddi. A T tube was inserted. The postoperative course was uneventful. The icterus rapidly cleared. Blood studies were normal. The T tube was removed four weeks after operation and the patient has remained asymptomatic for two months.

SUMMARY AND CONCLUSION

It is our impression that pneumocholecystitis is a fulminating type of actue cholecystitis, especially when occurring in a person with diabetes. The gallbladder walls are subject to greater tension when gas is present within the lumen and consequently there is an increased risk of gangrene and perforation. The tendency for the wall of the gallbladder to become necrotic and for gas to escape into the pericholecystic tissues, as frequently demonstrated in the reported cases, indicates a greater incidence of gangrene than in nongas producing infection.

Although no accurate evaluation of proper therapy is possible from the limited number of reported cases, it is our impression that early operation, with drainage or cholecystectomy would seem to be the method of choice to avoid abscess formation in the liver or peritoneal cavity.

There has been no previous record of a gallbladder being evacuated through a cholecystostomy wound. Although pathologic examination of the tissue in the case here recorded did not confirm the clinical impression that the material was the gallbladder, gross inspection of the tissue showed what appeared to be bile stained mucosa in small scattered areas with numerous bilirubin particles. At the second operation, no gallbladder was found, indicating that the gallbladder had sloughed through the cholecystostomy wound.

Pneumocholecystitis apparently occurs more frequently in males than in females. The published reports indicate that it is more common in a person with diabetes.

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INTERSIGMOID FOSSA HERNIA

CASE REPORT

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Swannanoa, N. C.

According to several investigators[3] the intersigmoid fossa is reported to exist in from 52 to 84 per cent of the population. It is very rarely of clinical significance except when the small bowel becomes incarcerated or strangulated in it. Twenty-seven cases of incarceration or strangulation have been recorded.[1, 2] In only 1 of these cases was the sigmoid involved.[1] When the fossa is present it usually is small, as is its opening which is directed downward. The small bowel is protected somewhat from it by the mesentery of the sigmoid which is to the right of the fossa.

FIG. 1. Roentgenogram of barium enema before operation shows abrupt obstruction to the flow of barium into the sigmoid.

From the Surgical Service, Veterans Administration Hospital, Swannanoa, North Carolina.

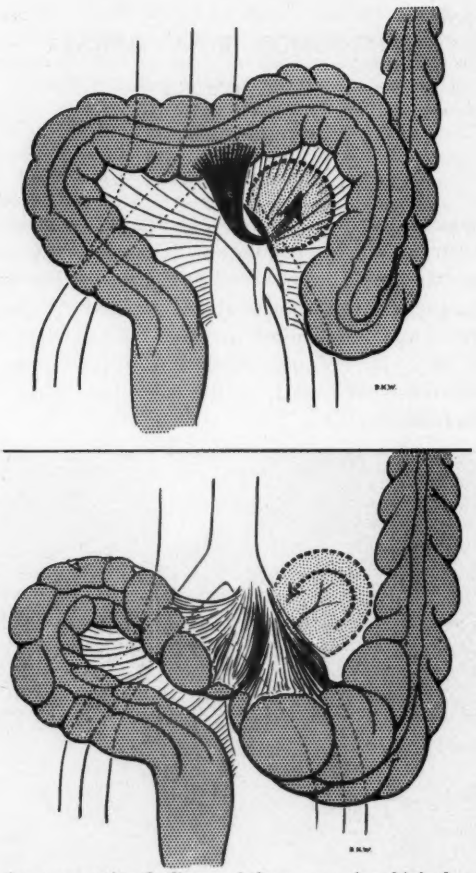

FIG. 2. Concept, from operative findings, of the manner in which the sigmoid became incarcerated in the intersigmoid fossa.

In most instances the diagnosis of intestinal obstruction was made, but the cause of the obstruction was suspected in only 2 cases prior to operation or necropsy. The mortality rate has been high. Aside from the rarity in which the intersigmoid fossa is involved in intra-abdominal hernias, this case is noteworthy in that it produced partial intestinal obstruction from incarceration of the sigmoid.

CASE REPORT

P. P., a 56 year old white man, was admitted to the Veterans Hospital, Swannanoa, North Carolina in March 1953, because of symptoms of benign prostatic hypertrophy and obstipation. He stated that he had been constipated for many years and his bowels would not move without taking laxatives. Several days at a time he would not have a bowel movement. He would lose his appetite; become nauseated and vomit. After he had taken

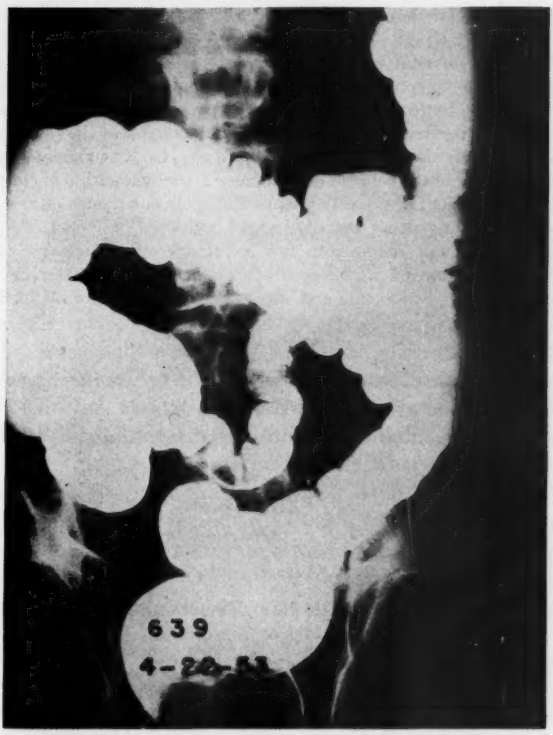

FIG. 3. Roentgenogram of barium enema after release of incarcerated sigmoid

several laxatives, the bowels would move and he would have diarrheal stools for several days. During these times his appetite improved and he felt well until it was necessary to resort to laxatives again to overcome the obstipation. Occasionally he saw a small quantity of dark blood and mucous mixed in the stools. He had had no episodes of fever and no weight loss.

Physical examination showed a slender, middle-aged, white man, quite deaf, who apparently was not in acute or chronic ill health. The abdominal examination was negative. By rectal examination the prostate gland was enlarged, boggy and tender. High in the rectum one could palpate an ill-defined moveable mass through the rectal wall. The laboratory data were essentially normal except for pus cells in the urine. The barium enema showed an obstruction to its passage in the midsigmoid where the barium stopped abruptly (fig. 1). The exact character of the lesion could not be determined by the roentgenologist who suspected that it might be the result of diverticulitis.

Operation was done on March 27, 1953. The abdomen was entered through a left lower rectus incision. The small and large intestines were examined. An intersigmoid hernia was found in a serosal pouch at the left side of the root of the mesosigmoid (fig. 2). Its opening was about 4 cm. in diameter. Its depth was approximately 5 cm. It was directed upward. The sigmoid loop was fixed in the hernial sac and there were rather dense adhesions of its mesentery to the parietal peritoneum which produced some angulation. The sigmoid was released and in doing so the mesosigmoid was perforated. This was repaired with intestinal

chromic catgut sutures. The hernial pouch was closed at its mouth with chromic catgut sutures.

The patient made an uneventful recovery. There was some improvement in bowel function, but he continued to be constipated and required mild laxatives. This apparently was due to changes which had occurred in the large bowel from partial obstruction of long standing. A barium enema following the release of the incarceration of the sigmoid showed some delay in the passage of the barium into the sigmoid (fig. 3). The sigmoid eventually filled well, as did the remainder of the colon. The film made after evacuation of the barium showed good emptying of the left half of the colon. Approximately one month later a transuretheral resection was done and the patient was discharged improved.

Six months later he was readmitted for treatment of a urinary tract infection. Proctoscopy and barium enema were negative. The only gastrointestinal symptom was constipation which was mild in nature and was relieved by taking mineral oil.

SUMMARY

Up to 1953 there were 27 cases of intersigmoid hernia described in the literature. With one exception all involved the small bowel.

A hernia of the sigmoid colon is here described. This is the second case of this type to be reported.

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FRACTURE OF THE FEMORAL NECK FOLLOWING PELVIC IRRADIATION*

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GENERAL CONSIDERATIONS

The purpose of this paper is to point out the relative incidence of fracture of the femoral neck following pelvic irradiation and that patients and their families should be cognizant of this complication.

Fracture of the femoral neck following pelvic irradiation is by no means a new entity. An excellent review of the literature was presented by Stampfli and Kerr in January 1947.[11] Fracture of the femoral neck following pelvic irradiation was first recorded in the literature by Baensch in 1927[1] and he added a second case in 1932. Philipp reported 4 cases in 1932.[9] Following these case reports several other cases were mentioned up until 1936 at which time the first report in the United States appeared in which Dalby, Jacox and Miller,[5] from the University of Michigan, presented 14 cases of fracture of the femoral neck following irradiation. One of the more recent presentations to appear in literature was in December 1953.[3]

In reviewing the literature the general consensus is that the etiology of fracture of the femoral neck following intensive pelvic irradiation is osteoporosis due to the irradiation. It has been demonstrated by Dahl[4] that osteoblasts were more sensitive to radiation than osteoclasts and he suggested that doses just large enough to inhibit the former may not affect the latter.

Much excellent material has been presented in the literature by Ewing,[6] Failla,[7] Slaughter,[10] Phemister,[8] and many others, regarding pathologic changes noted in tissues following irradiation. Vascular damage in the region frequently is mentioned and thought by some to be the important factor. Most cases have not shown vascular damage microscopically. If vascular damage were the predisposing factor it would seem that aseptic necrosis of the femoral head would be common. Actually this complication is not common following irradiation.

We have not had biopsy studies in our series, but from the radiologic standpoint osteoporosis invariably is noted. We believe that there is sufficient evidence to state that the etiology of fractures of the femoral neck in our cases is osteoporosis secondary to the irradiation. There were no cases of fracture due to metastasis.

Osteoporosis is one of the more conspicuous radiologic and microscopic findings. Boyd[2] stated that the two chief factors in the process of bony absorption are vascular absorption and osteoclastic activity. Vascular absorption plays the more important role and is brought about by the persistent dilatation of the

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TABLE I

Summary of cases

Patient	Age When X-Ray Treatment Started	Age at Time of Fracture	Interval in Months Between Treatment and Fracture	Femur Involved	Survival in Years With Carcinoma of Cervix	Type of Treatment for Fracture	End Result of Fracture	Remarks
1. GF	55	57	31	Right	9	Internal fixation	Satisfactory	
				Left		Internal fixation	Satisfactory	
2. BW	43	44	20	Right	3	Internal fixation	Satisfactory	
3. NL	41	44	33	Left	4	Internal fixation	Still on crutches 5 months postoperative	
4. IM	63	64	9	Right		Internal fixation	Excellent	
			43	Left	5	Nonweight bearing	Excellent	
5. MS	63	65	24	Right	13	Internal fixation	Excellent	This patient had more irradiation because of recurrence five years ago
6. MP	62	65	24	Right		Bed rest	Healed but walks with crutches	
			40	Left	8	Bed rest		
7. SM	65	68	23	Right		Nonweight bearing	Satisfactory	Not included in survival because incurable at start
				Left				
8. TC	66	67	15	Left		Nonweight bearing	Too early for evaluation	Carcinoma of ovary not included in survivals

Total and Averages of Cases

Number of Patients	Average Interval in Months Between Treatment and Fracture	Number of Fractures	Average Years Survival With Carcinoma of Cervix
8	29.3	12	7

vessels in the haversian and Volkmann's canals. Hyperemia is followed by widening of the canals at the expense of bone and, if absorption becomes very marked, rarefaction may result. Osteoclasts probably play a secondary part in the process and it is probable that decalcification first must occur before the osteoclast can exert its phagocytic action.

REVIEW OF CASES

During the past seven years, between Jan. 1, 1947 and Dec. 30, 1953, we have had 106 patients who have received intensive pelvic irradiation. Most of the patients have been followed in our tumor clinic, and in this group we have had 8 patients with fracture of the femoral neck. Four of these patients had bilateral fractures, giving a total of 12 fractures following pelvic irradiation (table I).

Six of the patients had carcinoma of the cervix, 1 had carcinoma of the ovary and 1 had hemangio-pericytoma—malignant type—of the uterus. The 6 patients with carcinoma of the cervix received not only irradiation therapy but also radium was applied directly to the cervix with an average dose of 6000 mg. hours. By the League of Nations classification the 6 patients with carcinoma of the cervix were stage II through stage III. All of these patients with carcinoma of the cervix have been seen in the tumor clinic within the past four months and none of them shows evidence of carcinoma. One of these patients is a 13 year survival from the time of her original diagnosis of carcinoma of the cervix. In the interval of the 13 years she also had carcinoma of the rectum, coronary occlusion with cardiac decompensation, gastric ulcer, local recurrence of carcinoma of the cervix with more irradiation, and bilateral fractures of the femoral neck. At the last clinic visit she was in fair condition and doing her own house work at the age of 76 years with no evidence of carcinoma.

These 8 patients with fractures of the femoral neck constitute an incidence of 7.54 per cent of the total 106 patients who received intensive plevic irradiation. Stampfli and Kerr[11] reported an incidence of only 0.87 per cent and Dalby, Jacox and Miller[5] an incidence of 2.1 per cent. During the past seven years there have been in our clinic a total of 42 fractures of the femoral neck in women due to all causes. Of this number 12, or 28.5 per cent, were in patients who had received irradiation therapy of the pelvis. The youngest patient was 44 years of age at the time of fracture and the oldest was 68 years of age. The usual average age of patients reported in the literature with all types of fracture of the femoral neck is 63.5 years. The average age in this series was 59.2 years.

In general, our patients, for pelvic irradiation, are treated externally about the pelvis. The treatment is directed to a total of eight fields as follows: left and right anterior pelvis, left and right posterior pelvis, left and right lateral pelvis and left and right ischial areas. Therapy is given in daily dosages to a total of 2000 to 3000 r to each of the first four fields mentioned above and 1600 to 1800 r to each of the lateral and ischial fields. The average total dose of irradiation is 14,000 to 19,000 r given over a period of 16 to 20 days.

SUMMARY AND CONCLUSIONS

A brief review of reports of fracture of the femoral neck following pelvic irradiation is presented.

It is the consensus of opinion that the etiology of fracture of the femoral neck is osteoporosis due to irradiation.

Eight patients with 12 fractures (4 bilateral) of the femoral neck are reviewed. This is an incidence of 7.54 per cent of 106 patients having received intensive pelvic irradiation. Of 42 fractures of the femoral neck in women due to all causes, 28.5 per cent followed pelvic irradiation. This study covers the past seven years.

In the 6 patients with carcinoma of the cervix, since the apparent survival rate is good, and the prognosis as far as healing of the fracture is good, there seems to be no indication to reduce the total dosage of irradiation therapy.

Since pelvic irradiation has gained wide usage every physician should be on guard, and patients should be aware that this complication may occur.

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UROLOGIC COMPLICATIONS OF SICKLE CELL DISEASE

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Hematuria and priapism have been fairly well established as complications of sickle cell disease. Harrison and Harrison[5] and Lund and associates[7] have presented studies of hematuria in sicklemia and have reviewed the literature. The latter recorded 7 cases, bringing the total of recorded cases of hematuria in sickle cell disease to 43. Since the incidence of this disease in the Negro race has been estimated to occur in from 7 to 13 per cent, hematuria in sickle cell disease probably is more common than realized, and many of the cases of unexplained hematuria in Negro patients reported in the past could have been due to this disease.

Campbell and Cummins[2] reviewed the literature and reported on priapism in sickle cell disease. They presented 5 cases of this complication, to be added to the 16 previously reported for a total of 21 recorded cases. In their series of 102 male sicklemics, 5 complained of priapism, an incidence of 4.9 per cent.

A review of the records of the Lake City Veterans Administration Hospital from April 1947 to April 1953 produced 13 cases of sickle cell disease. From September 1949 to April 1953, 8 of these patients were observed and treated by the author, 7 had gross hematuria and the eighth had priapism.

CASE REPORTS

Case 1. H. J. F., was a 26 year old Negro man who was admitted to the urology service on Sept. 23, 1949, complaining of gross hematuria. His first episode of gross hematuria occurred in September 1944, and lasted only one day. In January 1946, hematuria recurred, but stopped in a few days following cystoscopic examination. The hematuria has recurred intermittently since then, and the outstanding feature has been exacerbation or aggravation by hard physical exertion, especially in hot weather.

On examination, voided urine was grossly bloody, and prostatic fluid showed a 4 plus pus microscopically. The blood study showed 3,250,000 erythrocytes per cu. mm., a hemoglobin of 50 per cent, a prothrombin time of 16 sec. (100 per cent), a non-protein nitrogen of 28 mg. per cent and 430,000 platelets. A Kahn test was negative. A hanging drop slide showed marked sickling of red cells.

The treatment consisted primarily of sulfonamides, penicillin, and vitamin K. Cystoscopy revealed blood spurting from the left ureteral orifice. Retrograde pyelograms were made, and the left renal pelvis was irrigated with 1 per cent silver nitrate solution. By the next day, the hematuria had stopped, and did not recur prior to Oct. 10, 1949, when the patient was discharged.

Case 2. Z. W., was a 28 year old Negro man who was admitted to the urology service on Sept. 27, 1949, complaining of intermittent gross hematuria of one week's duration. In 1943, while in military service, he was hospitalized for 18 days because of a complaint of passing *blackish* and *reddish-looking* urine. He was also treated for urethral stricture during his military service, and again in 1948. He had gonorrhea in 1937, but denied syphilitic infection.

Examination on admission revealed gross hematuria and urethral stricture. A com-

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plete blood count showed 4,170,000 erythrocytes per cu. mm., 77 per cent hemoglobin, and 9,800 leukocytes per cu. mm. Moderate sickling of the erythrocytes was found. A chest roentgenogram and intravenous pyelography were normal. Serology was positive for syphilis.

Following admission, sulfonamides and penicillin were given. Cystoscopy was done on September 29, but traumatic bleeding from the vesical neck obscured vision. However, the hematuria subsided in a few days after using catheter drainage, intravenous fluids, and a blood transfusion. There was no recurrence of urinary bleeding prior to Oct. 12, 1949 when he was discharged. From April 8 to April 25, 1952, he was treated on the general surgery service for a bullet wound of the right upper chest. Laboratory tests at that time did not show hematuria or anemia. The serology still was positive for syphilis.

Case 3. S. L. B., was a 26 year old Negro man who was admitted to the urology service on Oct. 10, 1950 complaining of right flank pain and gross hematuria of one week's duration. In 1943, while in military service he had a similar episode of gross hematuria, and on cystoscopic examination at that time, renal bleeding from the left kidney was noted. Laboratory studies showed a microscopic hematuria, a normal blood count, negative serology, a non-protein nitrogen of 36 mg. per cent, an icterus index of 5.4, a prothrombin time of 13 sec., a normal A-G ratio, and a blood platelet count of 260,000. Marked sickling was observed in the erythrocytes. A chest roentgenogram was negative. The intravenous and retrograde pyelograms showed a minimal blunting and dilatation of the right superior calyx.

The treatment consisted of a combination of sulfonamides, aureomycin, and penicillin. The right flank pain and hematuria subsided prior to his discharge on Nov. 16, 1950.

On May 25, 1951 there had been no further gross hematuria. He had had occasional pains in the left abdomen. The blood count, bleeding time, coagulation time, platelet count, and urinalyses were all within normal limits.

Case 4. R. M. H., was a 27 year old Negro man who was admitted to the urology service on Feb. 26, 1952 complaining of gross hematuria for the preceding week. He gave a history of intermittent hematuria from 1943 to 1947. A left nephrectomy was done at the Philadelphia Naval Hospital in 1947. This was complicated by a left thrombophlebitis which was treated with dicumarol. In 1949 the hematuria recurred, and he was then informed at the Philadelphia Naval Hospital that he had *sickle cell anemia*.

Examination was essentially negative except for the left flank incisional scar, and a slight increase in the measurement of the left leg. Gross and microscopic hematuria were present on several urinalyses, 9 of which showed pyuria. The prothrombin time, icterus index, and serology were normal. Sickle cell determinations were 82 and 85 per cent. The blood count was as follows: erythrocytes 4.6 million per cu. mm., hemoglobin 16.1 Gm., hematocrit 48 per cent, and leukocytes 8,600 per cu. mm. The intravenous pyelograms and chest roentgenogram were normal, except for the absence of the left kidney.

He was treated with oral vitamin K and sulfonamides. The hematuria was not found on urinalysis daily from March 7 to March 11, 1952 at which time he was discharged.

Case 5. G. W. S., was a 38 year old Negro man who was admitted to the urology service on May 14, 1952 complaining of painless gross hematuria of six week's duration, without a history of any such occurrence previously.

Urinalysis on admission showed gross hematuria with 4 plus albumin. The serology was negative, the prothrombin time was 100 per cent, the platelets were 250,000, the clot retraction was normal, and the sickle cell test revealed 20 per cent sickling. A chest roentgenogram, and retrograde pyelograms were normal except for a mild left hydronephrosis. The blood count showed the following: erythrocytes 2.8 million per cu. mm., hemoglobin 5.3 Gm. and leucocytes 11,000 per cu. mm. The non-protein nitrogen was 26 mg. per cent.

The treatment consisted of several blood transfusions, sulfonamides, and antibiotics (penicillin in combination with oral terramycin). The hematuria persisted, but at the time of cystoscopic examination and retrograde pyelography on May 21, the urine coming from each ureteral orifice was clear. Following this, the hematuria stopped, and did not reappear up to the time of discharge on June 5, 1952.

Case 6. S. G., was a 31 year old Negro man who was readmitted to the orthopedic service on March 17, 1953 for the removal of an intramedullary nail from the left tibia. A urologic consultation was requested because of hematuria and frequency of urination. In 1945, while in military service, he was hospitalized for four months for these symptoms. It was also elicited that he frequently had had penile erections that were prolonged up to one-half hour in duration.

On July 8, 1952, he was first hospitalized here for a simple fracture of the left tibia and fibula. An intermedullary nail was inserted on July 10. Laboratory reports done at that time are of considerable interest. Five urinalyses were reported as containing varying quantities of erythrocytes, and leukocytes and albumin. On July 10, the blood count was within normal range, the serology was negative. On August 6, the non-protein nitrogen was 25 mg. per cent. A chest roentgenogram and excretory pyelograms were normal. A diagnosis of subacute pyelitis was made to explain the urinary findings. Penicillin was given from July 10 to July 22. Urinalyses on August 6, 7 and 11 were entirely negative.

On October 7 he returned to have his cast replaced. On October 9 the blood count was normal, but the urinalysis revealed 10 to 15 erythrocytes and 6 to 8 leukocytes per high power field.

Cystoscopic examination on April 23, 1953 failed to show any vesical pathology, but the urine was a brown color and turbid. Retrograde pyelo-ureterograms made then were normal.

Laboratory findings during the latest hospitalization from March 17 to May 2, 1953 were as follows: urinalyses on six occasions showed from 2 to 4 plus erythrocytes, and from a trace to 2 plus albumin. The blood count was normal; non-protein nitrogen was 35 mg. per cent; the icterus index was 7; the Kahn was negative and the sedimentation rate was 8 mm. Three cultures and smears of a concentrate of 24 hour urine specimens were negative for tubercle bacilli. A test for sickling of erythrocytes demonstrated 50 per cent sickling in 48 hours.

The patient was treated with gantrisin, penicillin, and methylene blue from March 19 to March 31 without any significant change in the hematuria. He was given a two week's leave from March 31 to April 14, 1953. Gantrisin, aureomycin, methylene blue, and penicillin were administered from April 21 to April 29. On April 24 the intramedullary nail was removed from the left tibia. Following this, he was given a three day pass on April 29, 1953 and failed to return.

Case 7. J. Q. W., was a 30 year old Negro who was admitted to the urology service on July 23, 1953 complaining of right flank pain, gross hematuria, and fever. He had two previous hospitalizations here, both of which are of considerable interest.

He was hospitalized on June 17, 1946 complaining of gross hematuria of two weeks' duration with pain in the left abdomen and left testicle. A routine blood count on June 20 showed 2,410,000 erythrocytes per cu. mm., a hemoglobin of 44 per cent and 6,500 leukocytes per cu. mm. with 65 neutrophils, 24 lymphocytes, 1 monocyte, and 10 eosinophils. Several urinalyses showed gross hematuria. The serology was negative, and the non-protein nitrogen was 21 mg. per cent. In spite of two blood transfusions, the hemoglobin had dropped to 23 per cent, and erythrocyte count was 1,500,000 per cu. mm. by July 1. Cystoscopic examination showed blood tinged urine coming from the left ureteral orifice. Intravenous and retrograde pyelography showed poor delineation of left pelvis and calyces, and dilatation of the proximal 5 to 6 cm. of the right ureter. On July 5, a left nephrectomy was done, but unfortunately, no record of the gross or microscopic pathology of the left kidney could be located. The discharge summary stated that prevention of exsanguination was achieved.

The patient was first seen by the author in February 1949, when he was rehospitalized from Oct. 15, 1948 to March 18, 1949, with an admission complaint of gross hematuria and pains in the right flank. Urinalyses showed gross blood. The blood count was normal. Retrograde pyelograms showed a stricture of the right urether about 6 cm. distal to the uretero-pelvic junction, with dilatation and redundancy of this proximal 6 cm. length of the right ureter. By Feb. 11, 1949, a urinalysis revealed clearing of the hematuria, and the complete blood count was still normal except for 6 per cent eosinophiles. The total blood

proteins, A-G ratio, non-protein nitrogen, Bence-Jones protein, and roentgenographic bone survey were all within normal limits.

The latest hospitalization, began on July 23, 1953 when the patient was admitted complaining of right flank pain, gross hematuria and fever, (100 F.). Examination revealed the well healed left iliocostal incisional scar, and tenderness in the right flank.

The significant laboratory findings were: urinalysis on July 23 showed a specific gravity of 1.010, 4 plus albumin, and gross blood. The blood count showed a 43 per cent hematocrit reading, 15.4 Gm. hemoglobin, and 11,500 leukocytes per cu. mm. with a normal differential. The icterus index, serology, prothrombin time, non-protein nitrogen, sedimentation rate and direct eosinophil count were within normal limits. Six urinalyses from July 27 to August 8 revealed from 2 to 4 plus albumin and 2 to 4 plus erythrocytes. Sickling of the erythrocytes was reported as 80 per cent in 72 hours, and 70 per cent in 32 hours on two examinations. On August 4, the Addis count was 2.1 million erythrocytes, and 500 leukocytes per cu. mm., and on August 6, 2 million and 1 thousand per cu. mm. respectively. On August 10 and August 12, the urinalyses failed to show any gross or microscopic hematuria, and only a few leukocytes. Five more urinalyses from August 14 to August 21 were negative for blood, and leukocytes varied from a few to 50 per high power field. The intravenous pyelogram made on August 19 confirmed the absence of the left kidney, and showed the dilatation of the proximal third of the right ureter. The middle third of the ureter was found to be displaced almost to the midline, and the diagnosis of a retrocaval ureter has been carefully considered.

The treatment consisted of penicillin, vitamin B_{12}, and crude liver extract given parenterally, with gantrisin, multivitamins, and methylene blue given orally. Two blood transfusions were given on August 14 and August 25 since the hematocrit and hemoglobin had fallen to 33 and 12 Gm respectively.

Surgical correction of the right ureteral stricture was recommended, but it was postponed by the patient.

Case 8. J. M. S., was a 33 year old Negro man who was admitted to the urology service on July 13, 1952 complaining of bouts of painful priapism for the past three and one-half months. He had noted prolonged uncomfortable erections for the past few years, especially at night. For the past three and one-half months, he had had almost constant painful priapism, in spite of treatment by his local urologist who used aspiration, saline irrigations, and dicoumarol.

He was a well developed and nourished, 33 year old Negro man who appeared to be in acute distress. The mucous membranes were pale. There was a systolic murmur at the apical area of the heart. The abdomen was normal. The penis was erect and the corpora cavernosa were tender and indurated. The prostate was slightly enlarged and boggy. No stricture of the urethra or residual urine was found on catheterization. There were varicosities in both legs.

The laboratory studies were reported as follows: Blood count (July 13, 1952) erythrocytes 2.9 million per cu. mm., hemoglobin 8 Gm., and leukocytes 13,150 per cu. mm. The differential count showed 64 neutrophils, 25 lymphocytes, 1 monocyte, 1 basophil, and 9 degenerated forms. There was 25 per cent immediate sickling of the erythrocytes noted. The prothrombin time was 16 per cent or 25 seconds, the sedimentation rate was 6 mm., the non-protein nitrogen was 36 mg. per cent and the serology was negative. On July 14, the blood count showed 2,950,000 erythrocytes per cu. mm., 8.8 Gm. hemoglobin, and 12,200 leukocytes per cu. mm. The blood smear showed target cells, sickle cells and polychromism. There was only a trace of albumin in the urine.

The day after admission he was given a pint of blood. This was repeated on the next three days for a total of four pints. On July 15 the hematocrit was 29, and on July 18 it was 31. The prothrombin time was 80 per cent (16 seconds); non-protein nitrogen was 32 per cent; icterus index was 21; the hemoglobin was 9.7 Gm.; the immediate sickling was 15 per cent and total proteins were 7.1 Gm. per cent (albumin 4.1, globulin 3).

It was decided to treat the patient with stilbestrol, 25 mg. daily parenterally from July 15 to July 22, then 25 mg. daily orally to August 15.

The blood transfusions were repeated on July 28 and August 5. On August 1, the hemoglobin was 10 Gm. and the hematocrit was 33. On August 6, the hematocrit was still 33, and the hemoglobin was 11.1 Gm., the prothrombin time was 100 per cent (15 seconds), the icterus index 26, and the sedimentation rate was 13.

From July 14 to July 17, there was little change in the priapism, but by July 17, the penis had become somewhat softer and more pliable. The patient was comfortable, and had slept well for the first time in three and one-half months. By July 23 the penis was relatively flaccid and the patient was asymptomatic. On August 8, he was discharged, to return in a month for follow-up.

Second admission. He was readmitted to the hospital on August 31. He had developed an acute recurrence of painful priapism, so severe that he would roll on the floor crying with pain. Re-examination revealed an erect, indurated, very tender penis, pale mucous membranes, and somewhat enlarged breasts with increased pigmentation around the nipples.

The blood count on admission showed 11.4 Gm. of hemoglobin, the hematocrit was 35, and leukocyte count was 14,400 per cu. mm. The urinalysis was negative except for 1 plus leukocytes and albumin. On several subsequent blood studies, the hemoglobin and hematocrit levels varied very little, and sickling ranged from 25 to 46 per cent. The serology was negative, the non-protein nitrogen was 27 mg. per cent, the clot retraction time was normal, as was the prothrombin time. A chest roentgenogram was negative. Several urinalyses continued to show a mild albuminuria, with considerable pus.

Immediately on admission, stilbestrol was prescribed, 25 mg. intramuscularly daily and also four times daily orally; plus heavy sedation. Over a period of several days, the penile pain and priapism slowly resolved. He was observed for two weeks after recovery, and since there was no recurrence, was discharged on a maintenance dose of 5 mg. of stilbestrol daily, to return in 30 days for a follow-up.

Third admission. He was readmitted on Feb. 17, 1953 to the surgical service because of an ulcer of the left lower leg. He had kept up the maintenance dose of stilbestrol, 5 mg. daily, without any recurrence of the painful priapism.

Laboratory findings. On this admission, the urinalysis showed 3 plus pus, and 1 plus albumin, a hemoglobin of 10.2 Gm., a hematocrit of 32, serology negative, 80 per cent sickling of red cells, and 15,800 leukocytes with 81 per cent neutrophils.

Priapism was not present, and the breast enlargement had subsided. There was an ulcer, 3 by 3 cm. in diameter, on the medial aspect of the left lower leg.

On March 10, bilateral high ligation of saphenous vein and stripping to the knee was done. On March 23, a vein ligation in the right calf was done. All incisions and the stasis ulcer healed, and he was discharged on April 1, 1953. He was kept on a daily maintenance dose of 5 mg. stilbestrol during his hospital stay, and he was instructed to continue this dosage. He will be seen periodically, especially for the purpose of reducing the dosage of stilbestrol.

DISCUSSION

The relationship of sickle cell disease to hematuria and priapism becomes more apparent as increased clinical observations of such cases are reported. The pathogenesis of hematuria in sicklemia is obscure.

Of the 7 cases here recorded, 2 (case 4 and 7) had been subjected to nephrectomy (both left), only to have hematuria recur from the remaining right kidney. Another (case 3) apparently had renal bleeding from the left kidney in 1943 and the right kidney in 1950. The findings in these 3 cause one to reflect very seriously upon the advisability of nephrectomy. In fact, in none of the 5 patients

who still had both kidneys was it found necessary to do a nephrectomy. However, Harrison and Harrison[5] found nephrectomy necessary in 4 of their 9 patients. This treatment was far from substantiated in the cases here reported, and in fact, these cases contradict their proposal that the renal hematuria results from some inherent defect found only in one kidney. Lund and associates[7] also have made substantially the same observations.

The hematuria subsided in all 7 patients after a variety of therapeutic measures. Repeated blood transfusions, antibiotics (mainly penicillin), sulfonamides (gantrisin) and methylene blue seemed most beneficial.

Following the line of reasoning suggested by Finkler,[4] as reported by Aaron and Robbins,[1] that male hormone therapy may have been the precipitating factor in their series of patients with priapism, it may be presumed that probably an excess of endogenous androgenic hormone plays an etiologic role in the priapism of sickle cell disease. The beneficial results of stilbestrol therapy, as reported by Campbell and Cummins,[2] and Dodson and Frohbose[3] also suggest this androgenic factor. Smith[8] recommended dicumarol therapy for priapism, but it had been of no value in the case here presented.

Kenigsberg and McGavack[6] have reported that the urinary output of 17-ketosteroids was increased by therapy with testosterone propionate. Our future investigation of all cases of priapism will include determinations of excretion of 17-ketosteroids.

SUMMARY

Urologic complications of sickle cell disease have been discussed. Seven patients with hematuria and 1 with priapism due to this disease are recorded.

In the Negro race, sickle cell disease should be considered a possible etiologic factor in hematuria or priapism.

Surgery for either complication is to be avoided until intensive conservative therapy has been employed. In my opinion, nephrectomy especially is unwise in these cases.

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METASTATIC CARCINOMA OF THE HYPOPHYSIS CEREBRI*

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Primary malignant hypophyseal tumors are uncommon. Metastatic carcinoma to this site is even less common, in fact it is so exceedingly rare as to excite considerable interest when discovered. In a survey of the literature occasional vague references to metastatic pituitary carcinoma were noted, but only a few authenticated reports were found, many of these being in the foreign literature.

In view of the unusual character of this metastasis, this single case report is thought to be warranted.

CASE REPORT

A 57 year old white woman entered the Robert Packer Hospital in February 1952 complaining of a lump in the left breast which had been present for about two months. The past history was insignificant and the routine laboratory studies were normal. A radical mastectomy was done for carcinoma of the breast. The tumor was a medullary adenocarcinoma and axillary node involvement was demonstrated. No other metastases were noted at this time. Surgery was followed by a course of roentgen therapy.

The patient did very well for two full years, presenting herself at the end of this period complaining of a persistent and severe headache. Intracranial metastases were suspected on the basis of increased intracranial pressure, some cerebellar signs and bloody spinal fluid. The patient deteriorated rapidly and died.

Autopsy revealed metastatic carcinoma in the cerebellum with massive softening and hemorrhage. There was no gross abnormality of the pituitary but on microscopic examination of this small organ, several cords and clumps of carcinoma were noted in the subcapsular area of the anterior lobe (fig. 1). Mitotic figures were numerous. The posterior lobe was not involved. Examination of the remaining organs of the body, including the original operative scar, failed to reveal any other gross or microscopic evidence of metastasis.

DISCUSSION

It is peculiar that metastases occur so infrequently to the hypophysis. Most metastases reported to date have involved the posterior lobe. We were able to find only two specific reports in the literature of metastatic carcinoma involving the anterior pituitary.[2, 11] Although several theories have been advanced, including an anatomic theory, a blood source and distribution theory and a hormonal theory, none the less the real reason for a preponderance of posterior lobe metastases remains a mystery. Diabetes insipidus may develop following metastatic disease of the neurohypophysis; several such reports are found in the foreign literature[10, 13, 9, 7] and one in the American literature.[8]

In Schwartz's case the entire gland was replaced by malignancy. Perry reported a chorioepithelioma in a 22 year old man metastatic to the neurohy-

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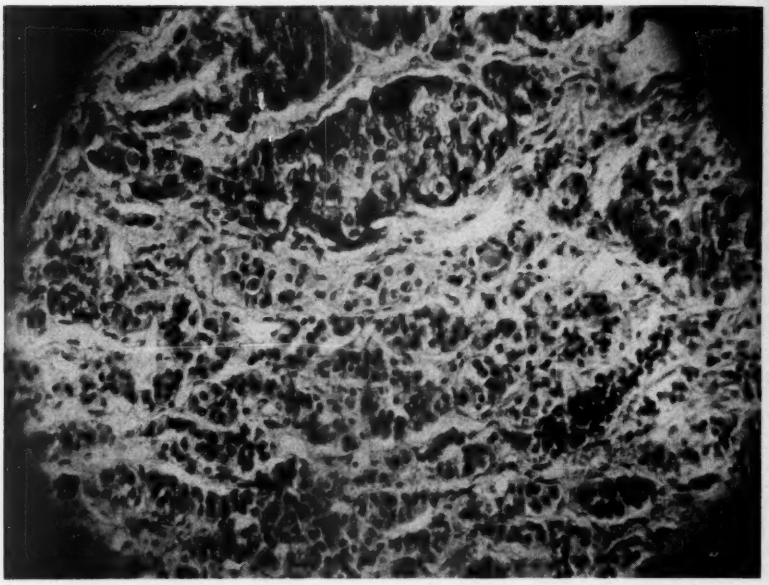

FIG. 1. Photomicrograph reveals metastatic breast cancer in subcapsular region. Adjacent to it is normal appearing pituitary. H & E, 220×.

pophysis. McCarthy and Karsner[6] contributed a case of thyroid metastasis in 1912 and there is one report of metastatic pancreatic carcinoma.[1]

Several cases of metastases from the lungs[4, 5, 12] were noted and Willis[14] points to the lungs as the most frequent source for this particular metastasis. The breast has been cited a couple of times. Ewing[3] pointed out that breast carcinoma is metastatic to the brain in about 4 per cent of cases, the cerebellum being the site of predilection.

SUMMARY

A most unusual and seldom recorded metastatic lesion is reported with the breast as the primary site. The metastasis was in the anterior lobe of the pituitary. The literature is briefly surveyed.

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ELEPHANTIASIS OF THE LOWER EXTREMITY

CASE REPORT

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In reporting the case of a patient with elephantiasis of a lower extremity we do not wish to claim or imply anything new in our method of treatment. In our opinion this presentation is justified because of some unusual features of the case, as well as the extreme involvement of the extremity.

Before proceeding with the case presentation a few remarks about elephantiasis in general might be in order, including a brief review of the etiology, pathology and other methods of treatment.

Elephantiasis is the late result of chronic, persistent lymphatic obstruction; if not congenital in origin, it is associated with a secondary infection, usually by streptococci. For many years elephantiasis has been intimately linked with filariasis and was thought to exist as a sequela to tropical infection. Today we know the specificity of filaria as an etiologic agent of this condition is doubtful even in tropical cases.

Dr. Rudolph Matas[4] in 1913 recognized that simple mechanical obstruction of the part without superimposed infection will not bring about the characteristic fibromatosis and other histologic changes which are peculiar to elephantiasis. His description of the underlying pathology was so accurate that nothing can be added to his original observations. There is marked thickening of the dermis and usually large areas of hyperkeratosis in the epidermis. There is marked dilatation of the lymph capillaries and small lymph vessels. These vessels may contain coagulated lymph. The subcutaneous tissue presents numerous swollen bundles of collagen fibers separated by great quantities of edema fluid. The sweat glands hypertrophy and the hair follicles disappear. The fat tissue is greatly replaced by fibrous tissue. There is a disappearance of the elastic fibers of the blood vessels and thickening of the vascular walls.

The treatment of choice for elephantiasis is one of prevention and control of the early progressive changes. Time does not permit going into detail concerning this treatment except to say that treatment must be active and vigorous, including strict bed rest with elevation of the limb, wearing an elastic stocking, antibiotics, plus general supportive measures.

The operative history of elephantiasis begins with attempts to scarify the tissues. In 1908 Handley[1] placed silk threads in the subcutaneous tissue in an attempt to re-establish lymph circulation. Following this fascia was used instead of silk threads. Then Lanz placed trephine holes in the femur since the bones are rich in lymphatic drainage. The results were all the same—nothing

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good happened. In 1912 Kondolean[2] described his procedure which consisted of an attempt to connect the superficial and deep lymphatic systems by excising large areas of the aponeurosis, thus allowing the subcutaneous layers to attach themselves directly to the underlying muscle. By this means new blood vessels and lymphatics supposedly would join the deep and superficial systems. The Kondolean operation was not entirely successful, but it represented the beginning of the thinking that eventually evolved the present method of attack.

Sistrunk,[5] in 1927, modified the Kondolean operation by removing a wedge of skin and a large wedge of subcutaneous tissue. Macey,[3] in 1940, placed a full thickness graft on the muscle fascia under the involved subcutaneous tissue and later excised the overlying elephantoid skin and subcutaneous tissue. Still later it was suggested that all of the involved skin and subcutaneous tissue be removed, with the fascia, and the denuded limb resurfaced with split thickness skin grafts. This was the method used in our patient.

A number of surgeons have performed this operation by removing the skin from the involved limb prior to excision of the underlying edematous tissue and utilizing this skin for resurfacing the muscle. This, of course, makes the operation easier for the patient and surgeon. Because of the rough, indurated skin in this patient we were unable to use it and had to obtain our grafts from other parts of his body.

CASE REPORT

The patient was a white man 46 years of age referred to us by Dr. Arthur Raynolds, Chief of Medical Service at the Veterans Administration Hospital, Bay Pines, Florida. He always had been in excellent health except for the swelling of his left leg which had begun approximately 16 years before. The swelling first appeared just below the knee in the calf of his leg and had become increasingly larger during the past 16 years, although there had been very little change in the size of his leg in the past five to seven years. He stated that his original weight, at the time the swelling began, was about 160 pounds, but for the past five years he had weighed from 300 to 305 pounds. He was able to walk on his leg with some difficulty, but had no significant pain. During the years he saw a number of doctors and several times was advised to have his extremity amputated. On occasions the skin had broken open and drained large quantities of clear fluid, but the areas always would heal spontaneously. He had never been out of the state of Florida except on one occasion, when he went to Atlanta to have his leg examined.

His physical examination was essentially negative except for his left lower extremity which appeared as shown in the accompanying photograph (fig. 1). Unfortunately the measurements taken of this leg were lost but, as can be seen from the photograph, his thigh was over 24 inches in diameter. The process extended from the groin and the subgluteal fold downward. There were deep creases at the knee and ankle. The foot showed marked increase in size. All of the dorsum of the foot and the toes were involved although the plantar surface of the foot appeared normal. Careful questioning revealed no episode of acute inflammatory disease involving this extremity previous to or at the time the swelling began to be noticeable.

He was admitted to the Georgia Baptist Hospital on April 24, 1952. At that time his blood Kahn was negative. The urinalysis and blood count were within normal limits. His blood pressure was 160/80. On April 25, after preoperative medication of $\frac{1}{6}$ grain of morphine and $\frac{1}{150}$ grain of atropine, he was taken to the operating room and given sodium pentothal with gas oxygen ether anesthesia. As his leg was being prepared, his blood pressure dropped rather suddenly to 80/55, although his pulse remained regular. Operation

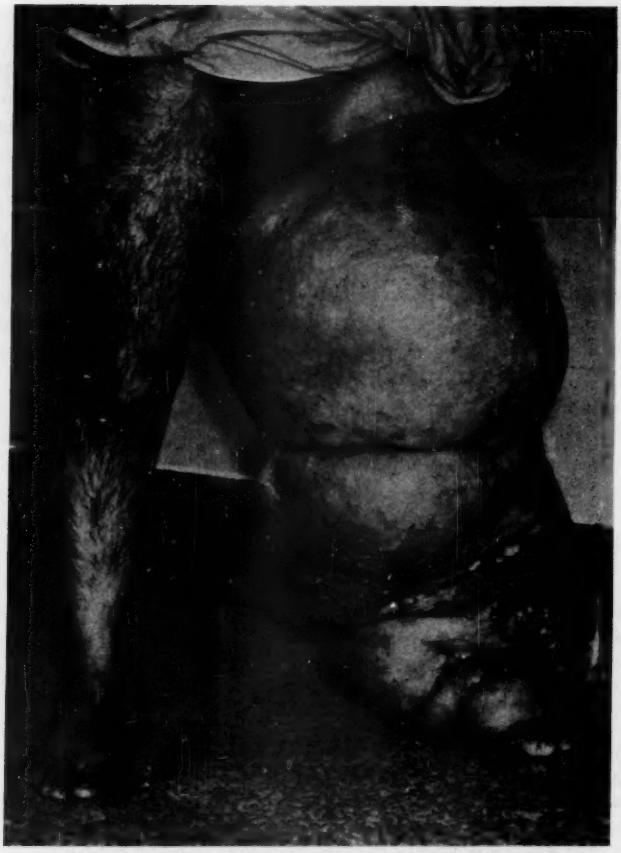

FIG. 1. Condition of lower extremity when first seen

was not begun immediately and he was given 500 cc. of blood intravenously. After about an hour his blood pressure had risen to 120/80. His first operation consisted of excising the involved tissue along the anterior surface of the thigh and leg, including a portion of the dorsum of his foot, down to the muscles which appeared grossly normal. When the tissues were incised, clear liquid literally *poured out* of the incision. Two suction machines were used to suck the excess fluid away and, during the course of this first operation, 7 gallons were sucked out of the wound and at least an equal or larger quantity was spilled on the floor. The weight of the tissue removed at the first operation was 47 pounds after the fluid had been allowed to drain out of it. The operation lasted for three hours and during the operation he was given three more transfusions of 500 cc. of blood each. His blood pressure remained stable throughout the operation and he was returned to the ward in good condition.

On May 9, he complained of sharp, stabbing pain in his left axilla. At that time his urine examination showed 4 plus sugar and blood sugar determination showed 312 mg. % Roentgenogram of his chest showed changes consistent with pneumonitis and pleuritis. He was seen by Dr. Bernard Wolff, an internist, who thought probably he had a septic

infarct in his left lung. The diabetes was controlled by diet and insulin for several weeks and then by diet alone.

On May 20 and 26, skin grafts were used to resurface the denuded areas and the grafts took nicely. On June 9, the involved tissue from the knee to the foot on the posterior surface was excised. During this procedure his condition fluctuated with, at times, a rather marked drop in blood pressure. At that operation he received a total of 1500 cc. of whole blood. Bleeding vessels were clamped and ligated and a vaseline dressing was applied. On June 30, split skin grafts were applied to cover the posterior surface of his leg and again the grafts took very nicely. The patient at that time showed some signs of discouragement and of becoming tired of the hospital. We accordingly gave him a brief respite from further surgery and let him go home for a short time. On September 30, he was operated upon again, at which time the involved tissue on the dorsum of his foot was excised down to the tendons and on October 6, that area was covered with a split skin graft. On October 21, the remaining edematous tissue was removed from the popliteal space up to the gluteal crease and the raw area covered with a split thickness skin graft.

At every operation the patient was given at least 1000 cc. of blood. He tolerated these last procedures very well and there was an excellent take of the grafts. He was dismissed from the hospital on November 2, at which time his skin donor areas had healed and the grafted areas were all healed except for a few small isolated ulcers.

The procedure was done in nine operative steps. All in all he received a total of 18 transfusions of 500 cc. each given at the times he was operated upon, and 14 transfusions of 500 cc. of blood at various times between operations. He showed some temperature elevation almost invariably after any operative step and at the time of his chest complication his

FIG. 2

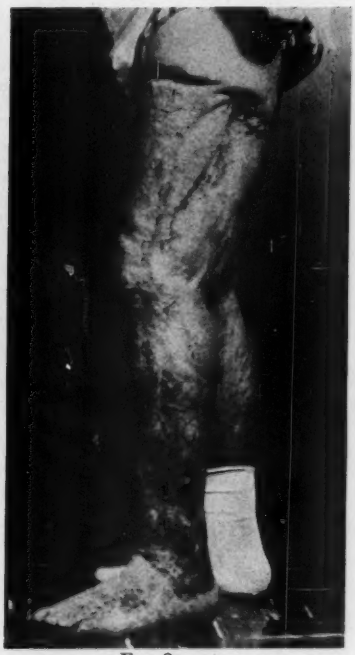

FIG. 3

FIG. 2. Anteroposterior view of lower extremity in July 1953
FIG. 3. Lateral view of lower extremity in July 1953

temperature rose to 104 F. only on one occasion. His last urine examination on Oct. 20, 1952, was negative for sugar and albumin and on the same date his blood showed 4,570,000 red blood cells per cu. mm. 8,950 white blood cells per cu. mm. and 13 Gm. of hemoglobin.

Microscopic examination of the excised tissue showed extremely thick, heavily keratinized skin surface beneath which there was a zone of dense fibrous tissue which was infiltrated with various types of chronic inflammatory cells. The findings were consistent with the diagnosis of elephantiasis.

The patient returned for examination on July 29, 1953, at which time the following note was made in his record: "Moderate swelling of lower leg with some pitting edema. There are two ulcers on the medial aspect of the ankle which have been present for some time. There is moderate scaling of skin grafts of foot and lower leg" (figs. 2 and 3). He is well pleased with the result of the operations and is able to wear his clothes and walk without a great deal of difficulty. A communication from him in January 1954 stated that his leg was still swelling a little, but he could avoid this by using an elastic bandage or stocking.

SUMMARY

A patient with extensive elephantiasis of his left lower extremity is reported. The possible etiology of this condition is discussed. He was treated by excision of the involved tissue and the application of split thickness skin grafts to the underlying muscle. We believe the results in this patient warranted the multiple operations and the time involved in his treatment. In our opinion, this represents the best treatment yet devised for extensive elephantiasis.

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EDITORIALS

THE RATIONALE OF MORE RADICAL SURGERY FOR BREAST CANCER

The prime objective of radical cancer surgery is the en bloc excision of the primary cancer together with its regional lymphatic drainage depots before distant metastases have occurred. *Early diagnosis* followed by *early more radical surgery* should increase the salvage rate of breast cancer provided the more extensive procedure is not accompanied by an increase in postoperative morbidity and mortality rates.

Cancer of the breast metastasizes primarily to two lymph node depots—the axillary and the internal mammary chain. In the average group of primary operable breast cancers approximately 60 per cent of the cases show axillary node metastases while the internal mammary nodes are involved in about 33 per cent of the cases. Other primary drainage routes which are less frequently involved are the intercostal lymphatics and primary hematogenous routes. In the great majority of cases which spread to the axillary or internal mammary nodes secondary drainage then extends into the nodes in the base of the neck. Here, the lymphatic vessels drain into the venous system at the confluence of the internal jugular and subclavian veins. When cancer cells enter this venous junction the disease becomes systemic and is beyond the scope of extirpative surgery.

In deciding where to limit the extension of radical surgery for breast cancer two problems arise: 1. At what stage in the extension of radical mastectomy does the possible added salvage obtained by more extensive resection of lymphatic bearing tissues become negated by increased operative morbidity and mortality rates and postoperative discomfort and disability? 2. At what stage in the progression of regional lymph node metastases does the disease become generalized and beyond the scope of surgical removal?

Furthermore, several conditions must be maintained if one is to profit from more radical surgical treatment of breast cancer.

1. The salient features of radical mastectomy should be preserved—en bloc resection, wide skin excision, meticulous thorough dissection of the axillary content, thin skin flaps—and, to this must be added the en bloc resection of other primary lymphatic drainage areas—and, in some cases, possibly secondary drainage areas as well.

2. The more extensive procedure should be applied only to patients whose general physical condition will tolerate the procedure and in whom one has reason to believe that the disease has not progressed beyond the regional lymphatics. Advanced cases with subclinical systemic metastases cannot be helped by more radical extirpative surgery.

3. The more extensive procedure must be planned so as to avoid undue post-

operative morbidity and mortality and should yield an adequate individual whose original general physical status in society is essentially unchanged.

4. The more radical surgery should be done only by competent surgeons with an excellent anesthetist in charge of the patient and with adequate postoperative care—otherwise postoperative morbidity and mortality rates will become prohibitive.

The first and most logical step to be considered in the extension of radical mastectomy is the inclusion of en bloc resection of the internal mammary lymph node chain—a primary lymphatic drainage depot. That this can be done without undue mortality and morbidity rates is attested by the 125 consecutive patients with primary operable breast cancer, treated by radical mastectomy in continuity with en bloc resection of the internal mammary lymph node chain at Memorial Center with but one postoperative death, and with an average hospital stay of eight days. Following surgery these patients are able to return to their normal level of activity. Thirty-nine per cent of these patients had metastases in the internal mammary nodes. Fifteen per cent of patients with negative axillary nodes had involved internal mammary nodes.

Further extension to include an in continuity low neck dissection is the next step to be considered. This already has been done as a practical surgical procedure. However, since this group of nodes comprises a secondary lymphatic drainage depot of the breast and lies in intimate relation to the entrance of the main lymphatic vessels into the venous system it is probable that the majority of patients with palpable metastatic cervical lymph nodes already have subclinical systemic metastases. One cannot expect a significant increment in salvage rate from this extension. In addition, this would be an extremely exhausting operation for both surgeon and patient. Nevertheless such an extension is feasible under certain limited circumstances. Certainly, it should be investigated. Further extension beyond the neck seems heroic and unlikely to succeed.

When a primary operable cancer is adherent to the chest wall—a not uncommon situation with lesions arising in the inframammary sulcus—the adherent chest wall should be excised en bloc in continuity with the overlying breast and pectoral muscles. This can be done without causing excessive disability provided the chest wall defect first is stabilized with a tense fascia lata graft to avoid paradoxical motion and then is covered by a sliding flap of skin and subcutaneous tissue.

Roentgen therapy and radical surgery are two proved tools in the treatment of primary breast cancer. They frequently must supplement each other and there are different situations in which one or the other may excel.

Early accessible cancer of the breast confined to the breast or with minimal involvement of the axillary and/or internal mammary nodes can be treated most adequately by radical surgical excision. Radiation therapy in this setting certainly will fail in a considerable number of patients who are not radiosensitive. On the other hand it is quite probable that in advanced cancers of the breast with extensive lymph node involvement and with spread to secondary lymph

node depots radiation therapy is less dangerous and is as likely to succeed as heroic radical surgery.

Rationally we can expect to salvage more cases of primary operable breast cancer through well-planned more radical surgery. However, we must await the test of time as the only true measure of the efficacy of such an attack.

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THE PRESERVATION OF THE BILE DUCTS

Whatever complications delay primary recovery, secondary operations on the gallbladder are a dreaded contingency. Accidental injuries in the biliary tract, particularly to the common duct, are the patient's chief hazard in every gallbladder operation. Here is an evil at all costs to be eschewed; yet, from cabinet rank to tourist class, the number of surgical cripples in our time is not diminishing.

The attempted repair of these hapless individuals occupies much of the surgeon's time and much space in the literature and current practice of gallbladder surgery. Scores of articles have appeared within the past decade on *how to repair* bile duct injuries, on the competing variations of this—at best—palliative measure. None will deny that in these sad cases the damage to the ducts is all too often irreprable.

The *prevention of injuries* to the common and hepatic ducts during operations upon the gallbladder, if less spectacular, is to my mind a subject far more important. Furthermore, it has not yet been fully explored. As teachers, and exemplars of a certain surgical success in some fields, we have failed dismally in this one. Too many surgeons, operating routinely upon gallbladders throughout the country, have never learned certain fundamental principles of safe biliary surgery: (1) that its *sine qua non* is the preservation of the bile ducts; (2) that the difficulties of doing a successful cholecystectomy in the acute stage of gallbladder disease may be extreme; (3) above all, that failure to identify the extrahepatic structure and its anomalies, coupled with reckless use of the hemostat, is inexcusable.

I speak gloomily; but it is the patient who pays for such fatal yet preventable disasters, while the responsibility for his tragedy rests on every senior surgeon and teacher of surgery. Particularly, in my opinion, the advocates of immediate operation for cholecystitis as a routine procedure may be accountable for some of the failures of their less accomplished followers. Not every surgeon is capable of excising an acutely inflamed and edematous gallbladder, the anatomic landmarks of which are both obscured and distorted. Furthermore, based on our experience with 1678 patients operated upon for cholecystitis at the Johnston-Willis Hospital, I am still convinced that only a small percentage of acutely diseased gallbladders demand or even justify immediate operation.

Teaching our students and surgical house officers how *not to injure* the common duct is, I think, a responsibility far more urgent than giving them a selection of any complicated repair service. In this behalf, I have drawn up a brief set of rules for the safety of the common and hepatic ducts and for the safety of the patient during a cholecystectomy.

1. Have a competent anesthetist.
2. Use an incision liberal enough to deal with abnormal as well as normal anatomy.
3. Recognize abnormal as well as normal structures.

4. Ligate the cystic duct *before* applying hemostat.
5. Ligate the cystic artery independently.
6. Do not try to complete a cholecystectomy on every patient; on the more difficult patients do a cholecystostomy or a partial cholecystectomy.
7. If unable to identify the cystic duct and cystic artery, always begin the dissection at the fundus.
8. Remember that the safety of the patient is more important than a completed cholecystectomy.

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BOOK REVIEWS

The editors of THE AMERICAN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The Editors do not, however, agree to review all books that have been submitted without solicitation.*

Rose and Carless' Manual of Surgery for Students and Practitioners. By SIR CECIL WAKELEY, BT., K.B.E., C.B., L.L.D., M.Ch., D.Sc., F.R.C.S., F.R.S.E., F.R.S.A., F.A.C.S., F.R.A.C.S. Fellow of King's College, London. President of the Royal College of Surgeons of England. Senior Surgeon, King's College Hospital; Director of Surgical Studies and Lecturer in Surgery, King's Hospital Medical School; Surgeon, Belgrave Hospital for Children and Royal Masonic Hospital; Consulting Surgeon to the Royal Navy; Examiner to the Universities of London, Glasgow, Durham, Sheffield, Wales and Ireland. Assisted by eighteen contributors whose names appear in the pages immediately following. Eighteenth Edition. With more than one thousand illustrations of which many are in color. The Williams & Wilkins Company, Baltimore, Md., 1952.

This text in surgery embraces all that is usually thought of as "general surgery" plus the surgical specialties and anesthesia. It is an old text having been published first in 1898, and this being the eighteenth edition. Consisting now of 52 chapters, there are substantial alterations since the previous edition. New chapters include the subjects of water and salt deficiency in surgery, biopsy in surgery, pathogenesis of infection, chemotherapy, disorders of the blood, blood transfusions, hemorrhage, and shock, the use of physical agents in surgery, burns and their treatment, and plastic surgery. This edition has been contributed to by 18 different authors. It is abundantly illustrated by 1,011 figures.

Discussion regarding any particular subject is meager, although perhaps this is compensated for by the wide and comprehensive range of subjects discussed. Practically no bibliography is present. To the undergraduate student, or the general practitioner, interested in general surgery from the English viewpoint, this book will serve as a beginning.

C. FREDERICK KITTLE, M.D.

Ulcerative Colitis and its Surgical Treatment. By BRYAN N. BROOKS, M. Chir., F.R.C.S. Reader in Surgery, University of Birmingham, Examiner in Surgery, University of Birmingham, Examiner in Surgery, University of Birmingham, Hunterian Professor Royal College of Surgeons, Assistant Surgeon, United Birmingham Hospitals. Foreword by F. A. R. Stammers, C.B.E., T.D., B.Sc., Ch.M., F.R.C.S., Professor of Surgery, University of Birmingham, Consulting Surgeon, United Birmingham Hospitals, E. & S. Livingstone, Ltd. Edinburgh and London, 1954. The Williams & Wilkins Company, Baltimore, Md. Price $7.50.

To those interested in diseases of the colon, whether they be operatively or mechanically inclined, this comprehensive monograph on ulcerative colitis will prove appealing and refreshing. In a pleasant and informative manner the author has combined a review of the pertinent literature with personal experience accumulated over many years, and from many patients with this disease, 58 of them having been subjected to some type of surgical procedure.

The book is divided into nine chapters which discuss the differential diagnosis, the pathology, complications, surgical procedures and ileostomy bag, preoperative management and indications for surgery, ileostomy, the management of an ileostomy, colectomy, and the general results in ulcerative colitis. Illustrative case histories are interspersed throughout to exemplify and reinforce the author's remarks and conclusions. The figures (89 of them) are numerous, of excellent quality, and contribute much to the format of the book.

The author is to be congratulated for his straight-forward approach, and his clear and illuminating exposition of this difficult subject.

C. FREDERICK KITTLE, M.D.

Atlas of Operative Technic: Anus, Rectum, and Colon. By HARRY E. BACON, B.S., M.D., ScD., F.A.C.S., F.R.S.M., F.I.C.S., F.A. P. S., Professor and Head of Department of Proctology, Temple University Medical School, Honorary Fellow, Royal Society of Medicine (England), Bordeaux and Abroise Pare Surfical (France), Madrid and Barcelona Surgical (Spain), Piedmontese Surgical (Italy), Venezuelan Surgical, Peruvian Surgical, Argentinian Surgical and Proctologic, Curitibia Surgical, Chilean Surgical, Brazilian Proctologic, and Dallas Southern Clinical Societies; Detroit Academy of Surgery; Diplomate, American Board of Surgery; Member, American Board of Proctology; and STUART T. ROSS, A.B., M.D., F.A.C.S., F.I.C.S., F.A. P.S., Attending Proctologist, Nassau Hospital, Mineola, New York, and Mercy Hospital, Rockville Center, New York; Assistant Attending Surgeon in Proctology, Meadowbrook Hospital, Hempstead, New York; formerly Assistant Visiting Surgeon, Kings County Hospital, Brooklyn, New York, and lecturer in Proctology, Polyclinic Medical School and Hospital, New York City; Secretary, American Protologic Society; Diplomate and Member, American Board of Proctology, Miembro Correspondiente extrajero Sociedad Proctologia de Argentina; Honorary Fellow, Brazilian Proctologic Society. St. Louis, C. V. Mosby & Company. Price $13.50.

In their preface, Doctors Bacon and Ross have stated that their purpose is to prepare an atlas of surgical procedures in proctology for the general surgeon and the occasional operator.

The volume is organized in two sections: Anorectal Operations and Colonic Operations. Preliminary to these two sections a short chapter on general considerations is given, including anatomy, preoperative and postoperative care, and anesthesia.

The most valuable part of this book lies in the type and quality of its illustrations, of which there are 403. They are done in pen and ink and are excellent, both technically and anatomically. The illustrations are accompanied by a facing page of concise step by step description of the various procedures. These are completely adequate.

This volume should occupy a prominent position in a surgical library, since it illustrates tried and accepted methods and procedures and well fulfills the purpose expressed by the authors.

THOMAS G. ORR., JR., M.D

BOOKS RECEIVED

Books received are acknowledged in this section, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

Collected Papers of the Mayo Clinic and the Mayo Foundation. Edited by RICHARD M. HEWITT, B.A., M.A., A.D.; A. B. NEVLING, M.D.; JOHN R. MINER, B.A., Sc.D.; JAMES R. ECKMAN, A.B., M.A., Ph.D.; M. KATHERINE SMITH, B.A.; CARL M. GAMBILL, A.B., M.D., P.P.H.; FLORENCE SCHMIDT, B.S.E.; AND GEORGE G. STILWELL, A.B., M.D. Volume XLV, 1953. Published June, 1954. Philadelphia and London, W. B. Saunders Company, 1954. Price $12.50.